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Academic Dissertation

Neuromuscular Research Center,
Department of Biology of Physical Activity,
University of Jyväskylä

UNIVERSITY OF JYVÄSKYLÄ

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ABSTRACT

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The present series of studies were designed to investigate the motor control mechanism during and after stretch-shortening cycle (SSC) exercise. Special emphasis was placed on stiffness regulation during SSC exercise. Neuromuscular mechanisms of the SSC action were studied in non-fatigued situations as well as in exhaustive, SSC exercise-induced fatigue conditions. In non-fatigue situations SSC performance can be characterized by a combination of pre-landing joint kinematics and post-landing muscle-tendon stiffness during the eccentric phase of the cycle. The centrally mediated pre-landing activity and eccentric action of the cycle seem therefore to play a role in regulating SSC. In the fatigue experiment, exhaustive SSC exercise induced delayed and slow recovery of performance and stiffness accompanied with delayed muscle damage, especially in the eccentric part of the cycle. Reduced and delayed stiffness was related to the damage-induced movement alteration of the SSC action. This may suggest that a pain-induced mechanism can have an inhibitory influence on the movement strategy. During SSC exercise, a great amount of alteration was observed in the pre-landing kinematics, which indicates greater sensitivity on the part of the pre-landing motor control strategy for the functional requirement of the peripheral system. Pre-landing ankle joint kinematics influenced post-landing stiffness and performance. In addition, alteration in post-landing stiffness was related to post-exercise delayed muscle damage. These interactions emphasize the chain reaction between pre-landing movement strategy, post-landing muscle mechanics and post-exercise performance. During the course of the SSC exercise, the neuromuscular adaptation process flexibly adapts across the joints and action phases of SSC. Consequently, the present series of studies indicates the possibility that the central nervous system maintains an optimum balance between the facilitation and inhibition of the peripheral system in terms of stiffness regulation during SSC exercise.

Key words: Stiffness regulation, neuromuscular fatigue, motor control, muscle damage, pre-activity

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LIST OF ORIGINAL ARTICLES

The present thesis is based on the following papers, which will be referred to by their roman numerals:

I. T Horita, P V Komi, C Nicol and H Kyröläinen 1996. Stretch shortening cycle fatigue: interactions among joint stiffness, reflex, and muscle mechanical performance in the drop jump. Eur J Appl Physiol 73: 393-403.

II. T Horita, P V Komi, C Nicol and H Kyröläinen 1999. Effect of exhaustive stretch-shortening cycle exercise on the time course of mechanical behaviour in the drop jump: possible role of muscle damage. Eur J Appl Physiol 79: 160-167.

III. T Horita, P V Komi, C Nicol and H Kyröläinen 1999. Interaction between prelanding activities and stiffness regulation of the knee joint musculoskeletal system in the drop jump: implications to performance. Eur J Appl Physiol, submitted.

IV. T Horita, P V Komi, I Hämäläinen and J Avela 1999. Exhausting stretch-shortening cycle (SSC) exercise causes greater impairment in SSC performance than in concentric performance. Eur J Appl Physiol, submitted.

V. T Horita, P V Komi, I Hämäläinen and J Avela 1999. Changes in neuromuscular performance during repeated stretch-shortening cycle (SSC) exercise I: adaptation patterns in the joint kinematics and electromyographic activities. J. Electromyogr Kinesiol, submitted.

VI. T Horita, P V Komi, I Hämäläinen and J Avela 1999. Changes in neuromuscular performance during repeated stretch-shortening cycle (SSC) exercise II: interaction between joint kinematics and time-varying stiffness regulation. J. Electromyogr Kinesiol, submitted.

CONTENTS

1 INTRODUCTION

In human natural locomotion such as running or jumping, muscle force is generated by a stretch-shortening cycle (SSC) in which a muscle is first stretched by an external force such as gravity or inertia of a moving limb followed immediately by a shortening contraction (Norman and Komi, 1979). As suggested by Komi (1992), the purpose of SSC is to produce more powerful concentric action as compared to the pure concentric action alone. In this regard, the pioneer studies of Cavagna et al. (1965 1968) have shown that the positive work was enhanced after the active stretch. This was demonstrated in the isolated whole frog muscle as well as in human muscle in vivo, and the mechanism for performance enhancement may involve the reutilizing of the stored elastic energy. Since of this elastic energy theory was proposed, many attempts have been made to identify further characteristic mechanisms of the SSC action in terms of elastic energy usage (e.g. Alexander and Bennet-Clark 1977, Asmussen and Bonde-Petersen. 1974, Aura et al. 1986, Cavagna and Kaneko 1977, Ito et al. 1983, Kaneko et al. 1984, Komi and Bosco 1978, Kyröläinen et al. 1990).

In addition to this pure mechanical advantage, the SSC action has an additional advantage with respect to the acute force gain induced by the stretch reflex in the initial lengthening phase, which was investigated by Liddell and Sherrington (1924) in the early 1900s. About 50 years later, in the human SSC action, the stretch reflex was first identified in hopping (Melvill Jones and Watt, 1971a) as well as in running (Dietz et al. 1979). Therefore, as suggested by Bosco et al. (1982a 1982b), the combined influence of both elastic energy and reflex potentiation may operate effectively during the SSC action.

In the neurophysiological sense, the stretch reflex is regulated by the neural circuit for the maintenance of postural balance and/or compensating for an external load (Hammond et al. 1956). Thus additional research interests have been directed at the "neuromuscular interaction (Granit 1958)" in natural locomotion including SSC. Granit (1958) wrote that "It has been found convenient to analyse these problems in terms of a concept such as loop gain, a derivative which is the amount of myotatic reflex tension (output) produced per unit extension (input) in grams per millimeter". This original concept of stiffness regulation was followed by the comprehensive study of Nichols and Houk (1976) about 20 years later. However, Engberg and Lundberg (1969) who were probably the first to investigate the stiffness regulation during natural locomotion,

including SSC, using a freely moving cat, by a combination of kinematics and electromyographic recordings. These authors suggested that stiffness regulation during the initial stretching phase is probably due to the proprioceptive reflexes while EMG activity as a whole is controlled by the central programming during SSC.

Since this animal model, the literature lacks attempts to report stiffness regulation as a part of the integrated motor control systems, including central preprogramming as well as peripheral muscular mechanisms in natural human SSC. However, technological advances developed in the past 10 years make it possible to investigate further the role of neuronal mechanisms during human SSC by H-reflex measurements during motion (Capady and Stein 1987, Dyhre-Poulsen et al. 1991, Moritani et al. 1990a, Simonsen and Dyhre-Poulsen 1999). In addition, there is an alternative approach, using the SSC fatigue model, which has been used in a series of experiments in the laboratory of Komi during the same period (Avela et al. 1998 1999b, Gollhofer et al. 1987a 1987b, Nicol et al. 1991 1996). Because muscle fatigue induces neural alterations mediated by reflex pathways (Asmussen and Mazin 1978, Bigland-Ritchie et al. 1986), one can suppose that SSC fatigue may induce alterations in stiffness regulation which are associated with modification of the neural activities which involve central preprogramming as well as local muscular contraction failure. The SSC fatigue model is a very powerful method and has consequently been used frequently during the past ten years (Komi 2000). The present study strengthened this SSC fatigue model to study neuromuscular fatigue mechanisms during exhaustive SSC exercise. A special emphasis was placed on stiffness regulation during SSC exercise.

2 REVIEW OF THE LITERATURE

2.1 Stretch-shortening cycle (SSC)

2.1.1 Neuromuscular basis of SSC

2.1.1.1 Force enhancement during stretch

The stretch-shortening cycle (SSC) type of muscle action is characterized by active prestretch immediately followed by concentric action (Komi 1984). One of the advantages of the SSC is that a greater force level can be achieved at the starting point of the concentric action as compared to pure concentric and isometric action. In human movement, this phenomenon has been demonstrated during arm pull (Asmussen and Sorensen, 1971), elbow flexion (Takarada et al. 1997b), squatting with barbell (Takarada et al. 1997a, Walshe et al. 1998), planter flexion (Svantesson et al. 1994), running (Cavagna et al. 1964) and jumping (Asmussen and Bonde-Petersen 1974). The fundamental mechanisms of these phenomena are based on the fact that the additional force enhancement can be obtained by the stretching of the active muscle, was first demonstrated by Gasser and Hill (1924) and Fenn (1924) followed by Abbott and Aubert (1952). Force enhancement during stretch depends on stretch velocity (Edman et al. 1978, Flitney and Hirst 1978, Sugi and Tsuchiya 1988) and stretch amplitude (Goubel 1987). The initial first force increment is due to the attachment of the cross-bridges between the actin and myosin filaments (Flitney and Hirst 1978), according to the cross-bridge model proposed by Huxley and Simmons (1971). Abbott and Aubert (1952) and Edman et al. (1978) further demonstrated the residual force enhancement after stretch, which decays very slowly (about 4 – 5 sec). A recent study (Edman and Tsuchiya 1996) has suggested that the parallel elastic elements which include passive structures of connectin/titin and nebulin (Maruyama et al. 1977, Wang et al. 1979) are responsible for the slow component of force enhancement as well as residual force enhancement after stretch with respect to sarcomere length heterogeneity (Morgan 1990). Thus force enhancement during the stretching phase consists of a contractile element (cross-bridges) as well as passive structures (connectin/titin and nebulin) in terms of sarcomere kinetics. However, a question might be raised regarding the

compatibility of the restricted *in vitro* results with those of intact human movement; however recent results in single fibres seem also to resemble those observed in the human muscles *in vivo* (Takarada et al. 1997b).

2.1.1.2 Role of stored elastic energy

It has been also proposed that the increase in mechanical energy associated with force enhancement during the stretching phase, which could be stored in the active elastic component (SEC) in series with the contractile element (CE), may recoil during the subsequent concentric phase and result in greater work output and/or improved efficiency (Hill 1950). Cavagna and Citterio (1974) and Cavagna et al. (1965 1968 1971) explained the work enhancement in the concentric phase of SSC as compared to pure concentric action by the recoil of the elastic energy stored in SEC. In this situation, a short coupling time between stretching and subsequent shortening is a prerequisite for the utilization of stored elastic energy (Cavagna et al. 1965, Thys et al. 1972, Bosco et al. 1981, Aura and Komi 1986) and the process may involve faster sarcomere shortening during the concentric phase of the cycle (Cavagna et al. 1994). The relatively higher mechanical efficiencies which are reported during walking, running, jumping and hopping (e.g. Asmussen and Bonde-Petersen 1974, Aura et al. 1986, Cavagna and Kaneko 1977, Bosco et al. 1987, Hof et al. 1983, Kyröläinen et al. 1990) could be explained by the recoil of the elastic energy. However Hill (1950), Cavagna and Citterio (1974) and Cavagna et al. (1965 1968 1985) did not distinguish between the active (cross-bridges) and passive (tendon) components of SEC, because they did not take tendon structure into account in their experimental model. Alexander and Bennet-Clark (1977) estimated the elastic strain energy stored in tendons to be 5 – 10 times higher as compared to that stored in muscles in human running. In line with this finding, Voigt et al. (1995) have shown the possible influence of tendon mechanical properties on the efficiency of the natural human SSC using the estimated tendon Young's modulus. Ettema et al. (1990) have shown the minor role of work enhancement by the CE during shortening as compared to the elastic recoil of the SEC using the whole muscle-tendon complex of rat gastrocnemius muscle. This result seems to suggest indirectly the possible influence of tendon structure on the recoil of the SEC. However, elastic energy storage in tendons has been also recognized as negligible because of the small length change in tendons (Jewell and Wilkie 1958, Cavagna et al. 1964). Baratta and Solomonow (1991), Roeleveld et al. (1993) and Hawkins and Bey (1997) have demonstrated continuous high stiffness in tendons within the optimum tension level that implies a simple force transmitting function of the tendon instead of that of storing elastic energy. In human SSC, the functional role of SEC and/or tendons has been evaluated mainly by the simulation model (Bobbert et al. 1986, 1996, Anderson and Pandy 1993) owing to the difficulties of the direct recording of the tendon stress and strain changes in vivo (Komi 1990). Furthermore, the fundamental concept of elastic energy is still controversial (Ingen Schenau 1984 1997). Therefore the mechanisms of the stored elastic energy and subsequent recoil in human SSC are in need of further explanation, perhaps by combining the direct recording of tendon force (Komi et al. 1996) with that of ultrasound techniques (Fukunaga et al. 1996).

2.1.1.3 Myoelectric activities including stretch reflex

In addition to the pure muscular component reviewed in the previous section, the SSC

action also involves neural circuits, in this regard the stretch reflex evoked via spindle afferent discharge due to the rapid stretch during the stretching phase (Melvill Jones and Watt 1971a, Dietz et al. 1979) plays an important role. The functional significance of the stretch reflex on the SSC is based on the fact that the significant force compensation by the short latency reflex can occur after force yielding due to the rapid stretch (Allum and Mauritz 1984, Hoffer and Andreassen 1981, Nichols and Houk 1976, Toft et al. 1991, Voigt et al. 1998). Human stretch reflexes are basically composed of three different components which are characterized as M1, M2 and M3 due to their latencies (Lee and Tatton 1978). However, in the reactive SSC action such as sprint running and rebound (drop) jumping, the short latency reflex has a dominant role because the contact time during ground is short and the stretch velocity high (Dietz et al. 1979, Dyhre-Poulsen et al. 1991, Gollhofer et al. 1990, Gollhofer and Kyröläinen 1991, Komi and Gollhofer 1997, Voigt et al. 1998). It has been also shown that blocking the stretch reflex by injection of a drug reduces SSC performance (Kilani et al. 1989). Therefore elastic energy and reflex potentiation may operate more effectively in the SSC action (Bosco et al. 1982b). Bosco et al. (1982a) have estimated the relative contribution of elastic recoil (72 %) and reflex potentiation (28 %) to total performance potentiation in the SSC as compared to pure concentric action.

Additional evidence suggest that the lengthening contraction induces selective recruitment of fast muscle and/or fast motor units (MU) (Nardone and Schieppati 1988, Nardone et al. 1989). Moritani et al. (1990a) have demonstrated further the selective higher H-reflex gain in the "fast" gastrocnemius muscle as compared to "slow" soleus muscle during maximal hopping. Fast twitch fibre possesses greater force development during stretch (higher stretch activation) as compared to slow twitch fibre (Galler 1994, Galler et al. 1994 1997). These factors are favorable to the more rapid generation of greater force in the SSC action. However as suggested by Komi and Gollhofer (1997), an effective SSC requires well-timed pre-activation before stretch, a shorter eccentric phase and shorter coupling between stretching and subsequent shortening as a longer action time diminishes the reflex potentiation. The elastic recoil of the mechanical energy in the SSC is also less if the coupling time is long (Cavagna et al. 1965 1994). Therefore pre-programmed activity and/or motor control strategy seems to be important for an effective SSC.

2.1.1.4 Motor control strategy

Moritani et al. (1991) have demonstrated preferential muscle activation between ankle extensor synergy, which involves pre-activation and eccentric phases, during hopping performed at different frequencies. This result indicates the existence of centrally programmed phase-dependent muscle activity combined with the stretch reflex activity. Dietz et al. (1979) have also characterized the electromyographic (EMG) activity during sprint running as the combination of centrally programmed pre-activity and superimposed proprioceptive stretch reflexes. In the drop landing, which corresponds to initial air-borne (dropping) phase during the drop jump used in an SSC study (Komi and Bosco 1978), programmed well-timed pre-activity has been shown according to the dropping height and landing strategies (Dietz et al. 1981, Melvill Jones and Watt 1971b, Greenwood and Hopkins 1976a 1976b, Dyhre-Poulsen and Laursen 1984, Santello and McDonagh 1998, McKinley and Pedotti 1992). Task-dependent H-reflex and stiffness modulations have also been found in the drop jump as well as in drop landing (Dyhre-Poulsen et al. 1991), and thereby further imply the possible influence of centrally

programmed activity. Because a natural SSC action such as running and jumping is usually operated by a multijoint movement, the coordination strategy within segments also influences the final performance of the SSC action. In jumping activity, foot placement at contact (Kovács et al. 1999), landing technique (Bobbert et al. 1987) and sequential joint extension (Alexander 1989, Hudson 1986) affect performance. In this connection, Dyhre-Poulsen et al. (1991) have found that the pre-landing H-reflex excitability is modulated depending on the drop jump technique. Therefore the preprogrammed activity and/or motor control strategy seems to have a influence on the subsequent reflex activity as well as on muscle mechanical performance, which could then lead to alterations in the elastic energy recoil during the contact period of the SSC action.

Regarding the multijoint control theory, biarticular muscles such as the gastrocnemius, rectus femoris and biceps femoris muscles play an important role in transporting the mechanical energy from proximal joint to the distal one as well as controlling the direction of force in a multijoint movement (Ingen Schenau et al. 1987 1992 1994, Doorenbosch and Ingen Schenau 1995, Bolhuis et al. 1998, Kumamoto et al. 1994). The Functional significance of the biarticular muscles has been confirmed in SSC exercise (Bobbert and Ingen Schenau 1988, Jacobs et al. 1993, Prilutsky and Zatsiorsky 1994, Prilutsky et al. 1998, Gregoire et al. 1984, Soest et al. 1993). Doorenbosch and Ingen Schenau (1995) have proposed the greater influence of sensory feedback on the biarticular muscle for fine-tuning of the force direction as compared to the mono articular muscle, which was in fact suggested about 30 years ago from a neurological point of view (Engberg and Lundberg 1969). However comparison of the neuronal regulation mechanism between mono and biarticular muscles has not been done yet. Consequently the neuromechanical basis of the SSC action involves many functional aspects that start from cross-bridge kinetics and go finally to the coordination of the whole movement by the central nervous system, in which one factor may be coupled with another factor. Thus additional research is required for a better understanding of the mechanisms of SSC exercise.

2.1.2 Training effect of SSC exercise

As reviewed in the previous chapter, the SSC action involves unique neuromechanical strategies. A series of training studies with rats conducted by the team of Goubel have found the unique adaptation patterns after SSC training (Goubel and Marini 1987, Pousson et al. 1991, Almeida-Silveira et al. 1994). Relatively greater increase in soleus muscle compliance have has shown after the SSC training (+16 %) as compared to endurance running (-5 %), strength training (+4 %) and squat jump training (+11 %) accompanied by a relative increase in fast twitch fibres. Because increased muscle compliance implies increased elasticity, SSC training may induce enhancement in the capacity of stored elastic energy even in "slow" muscle. In human SSC training, no significant alterations have been found in muscle histochemical properties (Harridge et al. 1998). However SSC training enhances SSC performance (Hewett et al. 1996, Kyröläinen et al. 1989) accompanied by an increase in eccentric EMG activity (Kyröläinen et al. 1989). Power-trained athletes who usually participate in SSC training have shown a higher reflex amplitude in the "fast" gastrocnemius muscle as well as greater force development compared to endurance-trained athletes (Kyröläinen and Komi 1994). These results demonstrate the specific effect of SSC training on explosive power production.

2.2 Functional significance of stiffness in SSC

2.2.1 Stiffness regulation

2.2.1.1 Stretch reflex

In the SSC action, when the muscle is subjected to a sudden change in length (stretching) by an external disturbance, muscle force is increased by the assistance of the stretch reflex to compensate to force yielding (load compensation) and/or postural balance. The ratio of force change to length change is called to "stiffness" (Houk 1979). As suggested by Granit (1958), stiffness regulation is a major concern for the neuromuscular integration of the motor control. Therefore stiffness regulation is believed to be an important factor with regard to the neuromuscular control of the SSC because initial high stiffness resulting from the short latency stretch reflex would lead to a higher force level during the eccentric phase (Dietz et al. 1979, Gollhofer et al. 1992). Yang et al. (1991) and Sinkjær et al. (1996) have demonstrated the significant contribution of the stretch reflexes on force production during the stance phase of walking.

In intrinsic muscle, stiffness is directly proportional to filament overlap and the number of attached cross-bridges (Flitny and Hirst 1978, Ford et al. 1981) so that a positive linear relationship can be observed between increased muscle stiffness and increased force level (Ettema and Hujing 1994). In intact muscle, stiffness consists basically of two components, intrinsic muscle stiffness and reflex mediated stiffness (Hoffer and Andreassen 1981, Nichols and Houk 1976). In the decebrated cat soleus muscle, the short latency stretch reflex improved the linearity of the muscle response (Nichols and Houk 1976), in which maximally a three-fold greater reflex stiffness was observed as compared to intrinsic muscle stiffness (Hoffer and Andreassen 1981). Reflex mediated stiffness showed a rapid and non-linear parabolic increase in gain with the increase in background force level (Hoffer and Andreassen 1981). In human muscle, a basically similar phenomenon has been observed but a difference may exist between agonist-antagonist pairs, in which case a higher reflex stiffness can be observed at the low force level in the extensor muscle (Sinkjær et al. 1988, Toft et al. 1991). The force gain from the reflexes corresponds to around 10 – 20 % of maximum voluntary contraction (MVC) force in the ankle extensors (Stein and Kearney 1995). Stretch reflex amplitude and force gains also depend on the amplitude and velocity of stretch (Allum and Mauritz 1984), co-contraction by the antagonist (Nielsen et al. 1994) and joint angle (Gottlieb and Agarwal 1978, Weiss et al. 1986). The combination of background force level, agonist-antagonist muscle and joint kinematics including stretch amplitude, stretch velocity and joint position should be considered to induce effective reflex gain. The stretch reflex is further influenced by previous muscle history (Gregory et al 1998, Wallace et al. 1998) and instruction before perturbation (Hammond et al. 1956, Sciarretta and Bawa 1990). Thus in the natural SSC action, external circumstances as well as pure muscular components may be considered to contribute to effective stiffness regulation. However, as the stretch reflex is simply followed by a proprioceptive feedback loop in the neural circuit, it is possible to speculate that the mechanisms of stiffness regulation involve the neural pathways with respect to length and tension control (Houk and Rymer 1981).

2.2.1.2 Length and tension control

Combinations of the excitatory and inhibitory neurons as well as feedback sensors regulate muscle length and tension in the spinal neural circuit. In general in change, muscle length is detected by muscle spindles located in parallel with the extrafusal fibres in which Ia afferents send the excitatory signal to the spinal cord (positive feedback). Muscle tension is sensed by the Golgi tendon organ located in series with the extrafusal fibres in which Ib afferents send a signal to the spinal cord via the Ib inhibitory interneuron (negative feedback). Some hypotheses have been proposed with respect to the length and tension control mechanisms involving this neural feedback circuit.

In 1953, Merton proposed the follow-up servo hypothesis in which movements are initiated by a command signal sent to fusimotor neurons (γ-motoneuron) innervating intrafusal fibres rather than skeletomotor neurons (α-motoneuron) innervating extrafusal fibres. However recent results do not support this hypothesis (Loeb 1984). The next hypothesis, introduced by Granit (1975), was called the length-servo assisted motion which was based on the α-γ coactivation. In this model the alpha and gamma motoneurons are activated simultaneously by the same command signal. The problem with this hypothesis was that no change occurs in the spindle afferents or feedback signal when the extrafusal and intrafusal motions are exactly the same. The third hypothesis was the stiffness regulation mechanism proposed by Houk (1979) in which spindle feedback is used in motor servo combination with the negative tension feedback from the Golgi tendon organs to maintain muscle stiffness. In this model, stiffness is considered to be a variable regulated by the intrinsic muscle property as well as by proprioceptive feedback which involves positive length feedback and the negative force feedback loop. This model has the merit that it allows a position with different degree of stiffness to be maintained (Loeb 1984). Therefore this model can be used in studying the stiffness regulation of SSC exercise. However, recent results stress the complexity of the spinal neural pathways, especially during fatigue which induces a decline in spindle support (Macefield et al. 1991), fusimotor activity (Ljubisavljević et al. 1992), feedback from the group III and IV afferents (Hayward et al. 1991) and thixotropic behavior of the muscle and muscle spindle (Proske et al. 1993, Hagbarth et al. 1985, Lakie and Robson 1988). Moreover, the possible existence of a skeletofusimotor neuron (β-motoneuron) innervating both the intrafusal and extrafusal fibres has been confirmed physiologically and anatomically in human muscle (Gandevia et al. 1986, Rothwell et al. 1990, Kakuda et al. 1998, Saito et al. 1977, Swash and Fox 1972). However, the functional consequence of the skeletofusimotor neuron in the spinal neural circuit in humans is remains unclear. Therefore additional attempts may be required to construct the whole picture of the neuromuscular integration.

2.2.1.3 Short range stiffness

When a muscle is forcibly stretched, two stages of force increment can be seen during the stretching phase both in an isolated muscle (Flitney and Hirst 1978) and in vivo human muscle (Hufshmidt and Schwaller 1987). An initial rapid increase occurred of about 0.2 – 1.2 % of the change in muscle length, which corresponds to 1 – 2 deg of change in the joint angle (Flitney and Hirst 1978, Hufshmidt and Schwaller 1987, Hill 1968), followed by a short plateau or decrement and then a secondary increase to the

subsequent force peak. The initial rapid increase was termed the "short range elastic component (SREC)" (Hill 1968) or "short range stiffness (SRS)" (Rack and Westbury 1974). A short plateau or decrement of force after the initial rapid increase was variously called the "elastic limit" (Hill 1968), sarcomere "give" (Flitney and Hirst 1978) and tension "shoulder" (Hufshmidt and Schwaller 1987). Short range stiffness was due to the initial stretch of the cross-bridges between the thick and thin filament and the subsequent "give" was due to the detachment of the cross-bridges (Flitney and Hirst 1978, Hufshmidt and Schwaller 1987, Hill 1968, Rack and Westbury 1974, Wood et al. 1993). Thus short range stiffness has been considered to be operative in the series elastic component of the cross-bridges (Joyce and Rack 1969). Because short range stiffness increases with the background tension level (Joyce and Rack 1969, Joyce et al. 1974), it has been suggested that short range stiffness is influenced by prior muscle tension and movement (Joyce et al. 1974, Morgan 1977). A recent study has further demonstrated the existence of fiber type specificity in short range stiffness (Malamud et al. 1996). Interestingly, short range stiffness has also been observed in the multijoint natural human movement, including SSC (e.g. McNitt-Gray 1991, Devita and Skelly 1992, Gross and Nelson 1988, Caster and Bates 1995, Dyhre-Poulsen et al. 1991, Farley et al. 1991, He et al. 1991, Gollhofer et al. 1992, Komi et al. 1987a). However, no information is available on the significance of the role of short range stiffness in the SSC action.

2.2.2 Stiffness evaluation from the engineering point of view

2.2.2.1 System identification approach

Because skeletal muscle has elastic as well as viscous characteristics (Levin and Wyman 1927) a system identification approach has been used to identify stiffness and viscosity during the single joint movement (Gottlieb and Agarwal 1978). In many cases, joint torque has been expressed as a combination of inertial, viscous and elastic components using the second order transfer function (Gottlieb and Agarwal 1978, Weis et al. 1986, Hunter and Kearney 1982 1983, Kearney and Hunter 1982, Flaherty et al. 1995). More recently, attempts have been made at the simultaneous and nonlinear identification of stiffness dynamics including possible neural feedback (Stein et al. 1995, Zhang and Rymer 1997). The results indicated asymmetrical response of the reflex action between the stretching and shortening contraction in addition to a relatively small contribution of reflex-mediated stiffness as compared to the increase in viscosity with an increase in the force level (Zhang and Rymer 1997). Although the important role of the stretch reflex has been recognized, the significance of the reflex action has been considered to depend heavily on the particular and/or experimental conditions (Kearney et al. 1997, Zhang and Rymer 1997). One of the technical limits of this method was that the estimated system could not follow the instantaneous change in time-varying stiffness in motion because the parameter estimation covers the whole time course of the movement in one series of equations.

As suggested by Kirsch et al. (1993), the stretch reflex has time-varying and task-dependent characteristics to regulate posture and movement. Therefore additional system identification was required. Bennett et al. (1992) have identified time-varying stiffness and viscosity during the voluntary cyclic elbow movement by the autoregressive moving average method coupled with ensemble averaging of 300 trials in which each parameter was identified at 1.67 ms intervals during the movement. Even

in such a simple movement (angular velocity; 2.1 rad·s^{-1}, range of motion; 1 rad, applied random torque; ± 4 N), time-varying stiffness changed instantaneously during the movement, showing a maximum difference of 6 – 7 times between minimum and peak values. This result clearly shows that stiffness is regulated instantaneously by the motor servo system during movement. Further attempts have been made to identify comprehensive stiffness regulation using the time-varying system of identification combined with the muscle activation model using the EMG signal under random perturbations (Kirsch et al. 1993, Kirsch and Kearney 1993 1997, Bennett 1994). Unfortunately, there was no attempt to apply these methods during the SSC action. However the time-varying system identification approach may offer useful information about stiffness regulation in SSC exercise.

2.2.2.2 Stiffness estimation by the oscillation technique

In running, jumping and hopping, the human body exerts a spring-like resistance against the external load (force), which induces oscillations within the system (body). It is possible to evaluate the spring stiffness and viscosity of the system by the engineering harmonic vibration theory by applying the instantaneous single impulsive vibration to the system. Cavagna (1970) first evaluated leg stiffness using the oscillation technique. Leg stiffness better explained the elastic energy stored during running (Cavagna, 1970). Because the oscillation technique can be used to evaluate the elasticity of the multi joint system as well as single joint system, it is convenient to use in evaluating multi joint SSC performance (Walshe et al. 1996, Walshe and Wilson 1997, Wilson et al. 1991b 1994), adaptability (Wilson et al. 1991a 1992, Newman et al. 1997, McNair and Stanley 1996) and fundamental stiffness regulation (Greene and McMahon 1979). However this procedure cannot be applied during the SSC action.

2.2.2.3 Stiffness estimation in SSC action

During sprint running or hopping, the ground reaction force curve shows a symmetrical bell-shaped curve with a short contact time lasting less than 200 ms (Cavagna et al. 1988, Dietz et al. 1979, McMahon et al. 1987). In this condition, the vertical ground reaction force-vertical displacement trajectory shows a linear relationship. Therefore the whole system (leg) can be expressed as a simple mass-spring system, according to Hooke's law, without viscosity (McMahon and Cheng 1990). Stiffness is calculated as the inclination of the force (F) - length (L) curve ($\Delta F/\Delta L$) and/or function of the natural frequency of the system (Cavagna et al. 1988, Blickhan 1989, McMahon et al. 1987, He et al. 1991). This method has been used for the investigation of the mechanics of running and hopping (Dalleau et al. 1998, Farley et al. 1991, Farley and Morgenroth 1999, Ferris and Farey 1997, Viale et al. 1998). However, this simple mass-spring system does not follow the longer action time of a SSC such as in the rebound jump, which has a relatively longer contact time (≈ 500 ms). The same could be the case in the non bell-shaped asymmetrical ground reaction force curve observed by Aura and Komi (1986) and Kyröläinen et al. (1990 1991). In this type of SSC action, the force-displacement trajectory does not show a linear but a non-linear relationship. Therefore the stiffness and viscosity of the system varies instantaneously with the possible voluntary support. In this case, the time-varying system identification approach seems to be appropriate for evaluating the stiffness regulation of the system. Dyhre-Poulsen et al. (1991) has demonstrated instantaneous stiffness change during hopping and landing.

However, this approach gives insufficient information as this model does not distinguish between stiffness and viscosity. Thus previous studies of stiffness regulation during the SSC action may be relevant to the specific type of SSC only.

In conclusion, as the review presented in this section shows there are several possibilities for evaluating stiffness in motion. Additional information regarding the characteristics of the measured stiffness can be found in the comprehensive review by Latash and Zatsiorsky (1993).

2.3 SSC fatigue

2.3.1 General features

As compared to fatigue studies conducted with isometric or concentric contractions, less information is available on SSC fatigue. Gollhofer et al, (1987a 1987b) were probably the first ones to investigate exhaustive SSC fatigue by the arm muscle SSC in which fatigue was induced by repeated submaximal rebound jumps (100 times, ≈ 6 min) on an inclined sledge apparatus. During fatigue loading, contact time increased progressively and this was confirmed by a later SSC fatigue study (Hortobágyi et al. 1991). The most pronounced finding was that the ground reaction force-time curve during SSC exercise and in the test jump after exercise showed an initial rapid rise accompanied by a sharp peak followed by a sharp drop in the force curve. In this connection, Komi et al. (1986), Nicol et al. (1991), Avela and Komi (1998) and Avela et al. (1999b) have shown a similar phenomenon after a long-lasting SSC, such as marathon running, accompanied by an increase in the action time of the SSC. These authors have suggested that the modulation of the force curve indicates the decreased tolerance of the initial impact with respect to the decrease in stiffness and subsequent deterioration in elastic recoil. This initial force modulation indicated alterations in the eccentric action of the SSC, especially with regard to the reflex response. A decreased stretch reflex response has been observed after SSC exercise of either short duration with rebound jumps (Nicol et al. 1996) or longer lasting marathon running (Avela et al. 1999b). Decreased eccentric EMG activity during the SSC has been shown in other types of SSC fatigue, such as hopping (Moritani et al. 1990b). Therefore the deterioration of the eccentric action as well as increased action time of the SSC is a common feature of SSC-induced fatigue irrespective of the duration of the exercise or type of SSC action. However, the stretch reflex was not changed after repeated maximal SSC exercise of short duration (≈ 60 s) accompanied by the potentiation of the contractile mechanism (Strojnik and Komi 1998). Thus different fatigue mechanisms may be operative between exhaustive susbmaximal and maximal SSC exercise. In addition, a different metabolic response has been shown between short and long-lasting SSC exercise. Short term exhaustive SSC exercise induces blood lactate accumulation immediately after exercise followed by a delayed increase in serum creatine kinase (CK) activity in contrast to long-lasting SSC exercise which induces a delayed increase in CK activity only (Nicol et al. 1996, Avela et al. 1999b). Because the delayed increase in CK activity has been considered to be an indirect marker of muscle damage (Schwane et al. 1983), possible muscle damage is also a common feature of SSC-induced fatigue (Komi and Nicol 2000).

2.3.2 Specific features

2.3.2.1 Muscle damage

When a muscle is subjected to unaccustomed work often accompanied by a lengthening contraction, delayed muscle soreness and/or muscle injury usually results (Hough 1901 1902). Eccentric contraction as well as SSC exercise has been shown to induce greater muscle injury accompanied by a force deficit as compared to shortening and isometric contraction (McCully and Faulkner 1985, Schwane et al. 1983). In humans, muscle injury has been further characterized by symptoms such as delayed increase in pain and soreness (Asmussen 1956, Komi and Buskirk 1972, Newham et al. 1983b, Jones et al. 1987), decrease in the relaxed joint angle (Jones et al. 1987, Nosaka and Clarkson 1995, Rodenburg et al. 1993, Clarkson et al. 1992, Howell et al. 1985, Stauber et al. 1990), increase in passive muscle stiffness (Howell 1993, Jones et al. 1987, Chleboun et al. 1998), altered force and position sense (Brockett et al. 1997) and swelling (Fridén et al. 1988b, Howell et al. 1985 1993, Nosaka and Clarkson 1995, Clarkson et al. 1992, Chleboun et al. 1998). In most cases, serum CK activity has been used as an indirect marker of muscle injury. This enzyme diffuses from the muscle intracellular compartments into the plasma as a result of the fiber necrosis caused by muscle injury (Armstrong et al. 1983, Schwane et al. 1983). However, caution should be taken in interpreting serum CK results with respect to the variability of individual response (Newham et al. 1983a) and lymphatic circulation in the removal of muscle protein (Stauber et al. 1990).

In general, similar morphological alterations have been found in muscles after pure eccentric exercise and, often, after SSC exercise. These changes are characterized as Z-band streaming and sometimes disruption, occurrence of abnormally shaped mitochondria, total disruption of the myofibrils, extracellular matrix disruption, alteration in the structure of the sarcoplasmic reticulum (SR), enlarged fibres by the inflammatory response, and loss of membrane integrity (Fridén et al. 1988a, Sjöström and Fridén 1984, Fridén and Lieber 1998, Warren et al. 1995, Byrd 1992, Stauber et al. 1990, Gibala et al. 1995). Therefore muscle injury induces alterations in myoplasmic contractile components as well as in cytoplasmic noncontractile components in addition to myotendinous junction (Asmussen 1956, Newham et al. 1983b, Tidball 1991). Such damage may preferentially occur in the fast twitch fibres (Jones et al. 1986, Fridén et al. 1983 1988a, Lieber and Fridén 1988, Sjöström and Fridén 1984). The fast twitch fibres possess narrow Z-bands (117 nm) as compared to the slow twitch fibres (156 nm), and may imply greater susceptibility to mechanical stress (Fridén et al. 1988a). Because the fast twitch fibres may be selectively recruited during the lengthening contraction (Nardone et al. 1989), the preferential use of fast twitch fibres in connection with this specific muscle architecture may explain the possible selective damage to the fast twitch fibres through eccentric as well as SSC exercise.

There are various mechanical parameters influencing the force deficit found during eccentric contraction, such as number of contractions, amplitude and velocity of stretch, peak tension during stretch and initial muscle length (Talbot and Morgan 1998). Talbot and Morgan (1998) have shown that muscle length, number of contractions and stretch amplitude have a strong effect on force deficit as compared to stretch velocity and peak tension during stretch. This result is in agreement with that of the study by Liber and Fridén (1993) in which muscle damage was not related to high force but to the magnitude of active strain. Brooks et al. (1995) have further shown that a single

stretch of only 30 % strain induces a significant force deficit. Thus initial mechanical injury plays an important role in the damage process. Mechanical injury also has an influence on sarcomere integrity. Higuchi et al. (1988) have demonstrated that the thin filaments did not re-enter the thick filament after extreme stretch. Because of the heterogeneity of sarcomere length along the fibre (Morgan 1990), the weakest sarcomeres are extremely stretched even in the normal stretching condition (Wood et al. 1993). Therefore, the altered sarcomere integrity associated with muscle injury would interfere with the smooth performance of the SSC action.

In Armstrong's (1990) summary the whole process of adaptation of exercise-induced muscular injury consists of four stages: (1) initial stage which includes the initial injury; (2) the autogenetic stage which includes the proteolytic and lipolytic systems which begin to degrade the cellular structures; (3) the phagocytic stage which involves the inflammatory response which begins 4 – 6 hours after the initial event and lasts 2 – 4 days after exercise; and (4) the regenerative stage, which covers regeneration of myofibres in the injured muscle and lasts about 4 – 6 days after the initial event. As a consequence, it takes about 10 – 14 days to fully repair the injured muscles. Faulkner et al. (1993) have proposed a bimodal sequence for the force deficit, using the mouse model in which the initial force deficit caused by the initial mechanical injury is followed by a subsequent secondary force decline mediated by delayed phagocytic activity. In this connection, MacIntyre et al. (1996) have shown a similar bimodal trend for force decline in humans. Interestingly, a similar phenomenon also has been found after long-lasting SSC exercise such as marathon running. Avela et al. (1999b) have demonstrated an initial deficit in the stretch reflex and reduction in peak force during the rebound jump followed by delayed secondary depression 2 days after exercise. Nicol et al. (1996) have shown that the delayed recovery of the stretch reflex and CK clearance are interrelated after exhaustive SSC exercise. These results indicate the possible influence of muscle damage on SSC performance, including the reflex neural pathway.

2.3.2.2 Possible link between muscle damage and neural pathway

Small group III and IV afferents have been activated by isometric contraction, muscle stretch, muscle fatigue, locomotion, lactate injection, mechanical stimulation and inflammation (Sinoway et al. 1993, Hayward et al. 1991, Pickar et al. 1994, Kaufman et al. 1983, Berberich et al. 1988, Schaible et al. 1983, Kniffki et al. 1978). It has been suggested that damage-induced pain and soreness might be transmitted by group III and IV afferents activated by sensitizing substances such as bradykinin, histamine, potassium and prostagrandins (Ebbeling and Clarkson 1989, Mense and Meyer 1988). In addition, these afferents have negative feedback on the α-motoneuron via the inhibitory interneuron (Cleland et al. 1982, Kniffki et al. 1981). It has been proposed that motoneuron firing rates can be regulated by reflexes from these small afferents in the fatigued muscle (presynaptic reflex inhibition) (Bigland-Ritchie et al. 1986). The decreased stretch reflex accompanied by a delayed increase in serum CK activities after SSC exercise seems to be partly due to presynaptic inhibition from the group III and IV afferents (Nicol et al. 1996, Avela et al. 1999b).

However group III afferent discharge also induces increased fusimotor discharge (Ellaway et al. 1982). Increased fusimotor discharge during and after the fatiguing contraction in the cat model also indicates the excitatory coupling between the fusimotor neuron and group III and IV afferents (Ljubisavljević et al. 1992). More

recently, this excitatory coupling has been confirmed in humans, in which experimental muscle pain without muscle injury increased the stretch reflex (Matre et al. 1998). On the other hand, disfacilitation of the α-motoneuron pool due to the decreased discharge of the spindle Ia afferent has been shown during sustained muscle contraction in humans (Macefield et al. 1991). Therefore it seems likely that the possible tradeoff between disfacilitation of the α-motoneuron and fascilitation of the γ-motoneuron during muscle fatigue may operate to optimize the balance between the autogenetic inhibitory and excitatory influence on the extrafusal fibres (Ljubisavljević et al. 1992).

Interestingly, both the stretch reflex and H-reflex depression have been shown after repeated long-lasting (1 hour) passive stretches in which simulated long-lasting SSC exercise was not accompanied by any increase in blood lactate and serum CK activities (Avela et al. 1999a). The H-reflex measurement has been used to evaluate the excitability of the spinal α-motoneuron pool (Schieppati 1987). It is known that the H-reflex and stretch reflex involve the same neural pathway in which the Ia afferent is mainly involved. However presynaptic inhibition via spinal interneurons through afferents with a large (Ia and Ib) and small (III and IV) diameter and/or central inhibition could influence the H-reflex as compared to the stretch reflex in which, through the spindle the Ia afferent is mainly involved (Schieppati 1987). Thus, the parallel decline in both the stretch reflex and H-reflex after passive stretches seems to exclude the possibility of selective presynaptic inhibition on the α-motoneuron pool as well as decrease in fusimotor support to the spindle. Therefore disfacilitation of the α-motoneuron pool is probably due to the decrease in spindle sensitivity related to the alteration in intrafusal fibre compliance after repeated passive stretches (Avela et al. 1999a). Because the stretch reflex as well as spinal excitability had recovered 30 min after passive stretches (Avela et al. 1999a), this result clearly demonstrates the thixotropic behavior of the muscle spindle as proposed by Proske et al. (1993). This implies that cross-bridge deformation occurs in the intrafusal fibres due to repetitive stretches. Possible intrafusal fibre damage has been also suggested (Komi and Nicol 2000). This may be also due to the fact that the position sense is significantly altered over 3 days after eccentric exercise (Brockett et al. 1997).

In addition, a significant reduction in the H-reflex / maximum M wave (H/M) ratio after exercise has been shown in either middle-lasting (20 min run) or long-lasting (marathon run) SSC exercise (Avela et al. 1999b, Bulbulian and Bowles 1992). Because the M wave indicates electrical response in the muscle (Enoka and Stuart 1992), the change in the H/M ratio after exercise represents a change in the excitability of the α-motoneuron pool. Therefore possible central fatigue after SSC exercise can not be excluded, as suggested earlier (Asmussen and Mazin 1978). Consequently, exhaustive SSC exercise alters neural functions drastically with respect to possible muscle damage, which may involve both extrafusal and intrafusal fibres.

2.3.2.3 Adaptation of the strategy during SSC fatigue

It has been shown that muscular fatigue alters the coordination of the multijoint movement (Forestier and Nougier 1998, Jones and Hanson 1971). Therefore the neuromuscular SSC adaptation pattern during fatigue is also one of the major concerns regarding the neuromuscular fatigue mechanisms of SSC exercise. Patla (1987) has proposed the neuromuscular adaptation model during sustained activity in which the adaptation process consists firstly of an alteration in neuromuscular strategy and secondly of alterations in task (movement) and neuromuscular strategy. The adaptation

of the neuromuscular system may have the following four stages: (1) on the motor unit (MU) level, (2) between MUs in a muscle, (3) between muscles across a single joint, (4) between muscle across multi joints (Patla, 1987). In this connection, Bonnard et al. (1994) have shown the the tradeoff mechanism between muscles at the ankle joint and tradeoff across the joints (knee – ankle) during fatiguing repetitive hopping (n = 4, average 35 min). However, the neuromuscular adaptation process during running or jumping in which larger muscle groups are recruited to perform a more complicated multijoint SSC action as compared to single joint hopping has not yet been investigated. Only limited information is available on kinematic changes at several points during running events (Nicol et al. 1991, Bates et al. 1977, Elliot and Ackland 1981, Elliot and Roberts 1980).

It has been shown that prolonged repetitive stretches induce a dramatic alteration in the viscoelastic properties of the muscle (Rack et al. 1983) in terms of the thixotropic behavior of the muscle as well as of the muscle spindle (Proske et al. 1993). In addition, extreme stretches induce deterioration in the sarcomere kinetics during contraction (Higuchi et al. 1988). Consequently, exhaustive SSC exercise induces potent changes in the peripheral machinery. As reviewed in the previous chapter, change in muscle stiffness is coupled with the previous muscle contraction and/or movement (Morgan 1977, Joyce et al. 1974). Therefore, the system as a whole is probably required to modify the movement strategy adapted to maintain stiffness during fatiguing SSC exercise. Thus investigation of the precise neuromuscular adaptation mechanisms during multijoint SSC exercise may offer useful information for basic and/or practical research on the natural type of SSC action.

3 THE PURPOSE OF THE STUDY

The neuromuscular basis of the SSC is well documented in terms of muscle mechanics as well as reflex interaction. In terms of the basic motor control aspects, many studies have investigated stiffness regulation in the simple single joint movement during non SSC action. So far, the literature lacks studies which examine stiffness regulation during multijoint SSC action. Therefore the control mechanisms of the multijoint SSC action are still unknown. After SSC fatigue, possible muscle damage has been demonstrated. The influence of muscle damage on the reflex pathway and on SSC performance has been also discussed. However, the neuromuscular adaptation process during SSC exercise has not been resolved in terms of stiffness regulation. Because muscular as well as neural functions might be altered during fatiguing SSC exercise, the motor control strategy regarding stiffness regulation seems to adapt flexibly to the alteration of the functional requirements of the peripheral system during exercise. Thus the general purpose of the present study was to identify the stiffness regulation motor control mechanisms operating during SSC exercise fatigue model. The detailed aims of the present study were as follows:

1) In landing activity, pre-programmed pre-landing activity plays an important role. In addition, stiffness regulation during the contact phase of the SSC would regulate power production during movement. However no attempt has been made to examine the interaction between pre-landing activity and subsequent stiffness regulation during movement. The purpose of the first part of this project was to clarify the neuromuscular factors which regulate SSC performance with special reference to pre-landing activity and subsequent stiffness regulation during the contact phase of the SSC action (III).

2) It has been demonstrated that exhaustive SSC exercise induces a decrease in SSC performance. Because an important function of the SSC action mainly relates to the eccentric phase, it might be assumed that SSC fatigue has a specific effect on the eccentric action. The purpose of the second phase was to identify the specific fatigue effect of exhaustive SSC exercise on the subsequent performance in comparison with the SSC action and pure concentric action (IV).

3) Exhaustive SSC exercise induces depression of the SSC action as well as delayed possible muscle damage. Therefore it could be proposed that SSC fatigue induces a possible alteration in stiffness regulation in the post-exercise SSC action. In addition, this alteration seems to be related to post-exercise delayed muscle damage. The purpose of the third study was to identify how exhaustive SSC exercise modifies stiffness regulation during the post-exercise SSC action (I).

4) It has been known that muscle damage induces a bimodal adaptation pattern in which the initial mechanical injury is followed by a secondary injury caused by the inflammatory response. This result seems to suggest that the initial mechanical depression might be sequentially connected to the subsequent change in performance during the follow up period after exhaustive SSC exercise. To confirm this, a study was undertaken to clarify the time course of the mechanical behavior of the SSC action, including stiffness, during the follow up period after the exhaustive SSC exercise in relation to possible muscle damage (II).

5) Neuromuscular function is altered during exhaustive SSC exercise. In this case, the motor control strategy seems to be modified to meet the functional requirement of the peripheral system, especially in maintaining the stiffness of the system. Therefore, possible compensation may be apparent in the progress of SSC fatigue. Thus studies were undertaken to identify the interaction between preprogrammed motor control and post action activities in relation to joint kinematics and stiffness regulation in the course of exhaustive SSC exercise (V and VI).

4 RESEARCH METHODS

4.1 Subjects

A total of 37 subjects participated in these studies. The subjects were well motivated physical education students. They were well informed about the possible risks associated with the experiment. During the experiments, the subjects were not allowed to practice any other physical activities to avoid possible influence on the results obtained. The physical characteristics of the subjects are presented in Table 1. These studies were approved by the University Ethics Committee.

TABLE 1 Physical characteristics (mean ± SD) of the subject groups.

	Experiment 1 (N = 9)	Experiment 2 (N = 10)	Experiment 3 (N = 10)	Experiment 4 (N = 8)
Age (yr)	28 ± 4	28 ± 4	28 ± 4	29 ± 4
Stature (cm)	180 ± 6	180 ± 4	180 ± 6	181 ± 4
Weight (kg)	78 ± 9	76 ± 5	78 ± 9	77 ± 5
Original paper	III	IV	I · II	V · VI

4.2 Experimental design, testing procedures and analysis

Four separate experiments were performed in this study. The first experiment consisted of measurement of drop jump (DJ) performance, including joint moment, joint stiffness and the mechanical DJ muscle model (III). The second to fourth experiments used a similar fatigue protocol inducing exhaustive SSC fatigue by the sledge apparatus. The second experiment compared SSC performance and pure concentric performance after exhaustive SSC fatigue (IV). The third experiment measured the stretch reflex, joint

moment, joint stiffness during DJ and its relation to possible muscle damage after SSC fatigue (I). In addition, the time course of the changes in the mechanical parameters of DJ as well as muscle damage after SSC fatigue were measured (II). The fourth experiment measured the time course of the kinematics and time-varying stiffness during exhaustive SSC exercise and its relation to subsequent SSC performance (V and VI). Table 2 summarizes the experimental design.

TABLE 2 Experimantal design of the present study.

	Experiment 1	Experiment 2	Experiment 3	Experiment 4
Drop jump	*		*	
Stretch reflex (drop jump)			*	
Max SSC (sledge)		*		*
Max SJ (sledge)		*		
SSC fatigue (sledge)		*	*	*
Joint moment	*	*	*	
Joint stiffness	*		*	
Mechanical Muscle model	*			
Time – varying Leg stiffness				*
Blood lactate CK		*	*	*

4.2.1 DJ test (I, II, III)

The subject performed DJ and counter movement jump (CMJ) on a force platform (60 × 125 cm, natural frequency: 200 Hz). The dropping height was set at 50 cm as high efficiency has been observed around 50 cm high in DJ (Komi and Bosco 1978). In the DJ trial, the subject was asked to rebound as quickly as possible with maximal effort just after ground contact. The subjects kept their hands on their hips throughout the whole movement to minimize hip extension. The duration of the flight phase (flight time: t_{air}) was obtained from the vertical ground reaction force signal. Take-off velocity (Vt) was considered to measure DJ performance, which was calculated from the flight time (t_{air}) according to the formula of Komi and Bosco (1978).

$Vt = 0.5 \cdot t_{air} \cdot g$

in which g = gravitational acceleration (9.8 $m \cdot s^{-2}$)
The DJ test was also used after the SSC fatigue measurements (I, II)

4.2.2 Sledge exercise (I, II, IV, V, VI)

4.2.2.1 Exhaustive SSC fatigue induced by sledge exercise (I, II, IV, V, VI)

In the present study, fatigue was produced by an exhaustive stretch-shortening cycle (SSC) exercise using a special sledge apparatus (Fig 1). This apparatus consisted of a 33 kg sledge which glided on a track inclined at 23 degrees from the horizontal. The subject sat on this sledge and was dropped from a pre-determined height along the track. As soon as the subject's feet contacted the force plate placed perpendicularly to the track, he jumped along with the track. The details of this apparatus have been described in other reports from our laboratory (Kaneko et al. 1984, Komi et al. 1987b, Aura and Komi 1986). Before the fatigue exercise, the maximal rebound height (100%) of each subject was determined on the sledge using a series of maximal single SSC, in which the subject was dropped from progressively increased heights (70, 80 and 90 cm). The fatigue exercise consisted of bilateral submaximal repeated SSCs with the lower limb on the sledge apparatus. Submaximal SSC load (rebound height) was set at 70% of the single maximal rebound height on the sledge in a pre-fatigue trial. The subjects were asked to reach the same submaximal rebound height throughout the exercise and instructed to perform as many repetitions as possible. Oral feedback was given to help the subjects to maintain the predetermined rebound height. The exercise was stopped when the subject could no longer reach his submaximal rebound height. In connection with the fatigue experiments, blood samples were taken to analyze lactate and serum creatine kinase (CK) activity immediately before and after exercise as well as 2 days and 4 days after exercise.

4.2.2.2 Jump test using the sledge apparatus before and after SSC fatigue (IV, V, VI)

The maximal drop jump was performed on the sledge apparatus from a predetermined height (Max SSC), so that the lower limb extensors are actively pre-stretched after touch-down followed by a concentric contraction (IV, V, VI). The maximal squat jump (Max SJ) was performed from a squatting position with the knee bent with no active pre-stretch, in which the extensor muscles performed pure concentric contraction (IV). Jump tests on the sledge are demonstrated in Fig. 1. The experimental protocols used in the fatigue experiments are summarized in Table 3.

TABLE 3 Experimental protocol in each fatigue experiment. Circle denotes the measurements after the SSC fatigue.

	Before	Exhaustive SSC Fatigue	0 min	10 min	20 min		2 hours		2 Days	4 Days
Exp. 2	Max SSC Max SJ		O	O	O O		O O		O O	O O
Exp. 3	DJ		O		O		O		O	O
Exp. 4	Max SSC				O		O		O	O
Blood	LA CK		OO O		O O		O		O	O

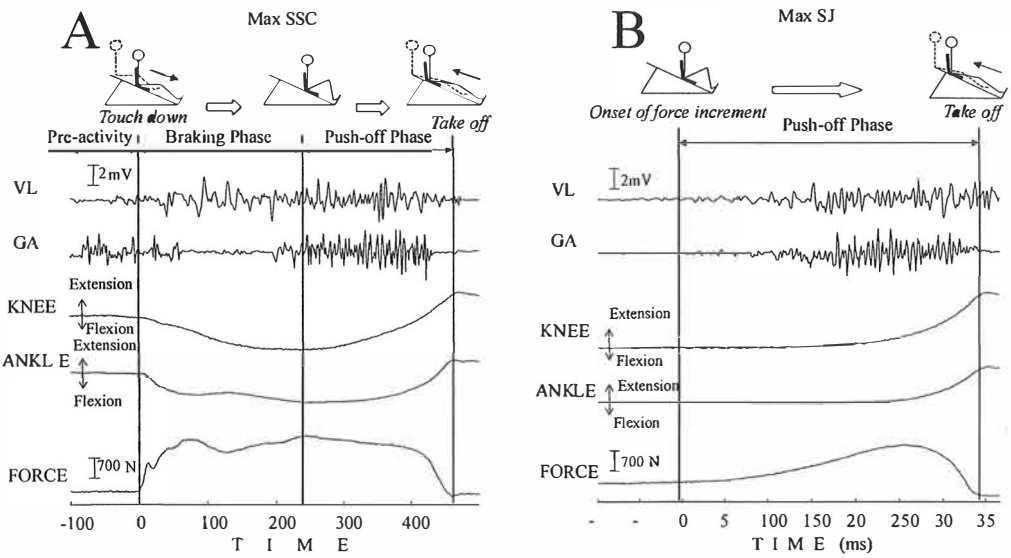

FIGURE 1 Examples of the records during Max SSC (A) and Max SJ (B) in one subject. *From top to bottom*, schematic drawing of the motion on the sledge apparatus, vastus lateralis (VL) and gastrocnemius (GA) electromyographic (EMG) activities, knee and ankle joint angles and force signal. The three vertical lines in Max SSC indicate the separation of pre-activation phase, braking phase and push-off phase, respectively. The Max SJ is started from the same position as shown at the turning point between braking-phase and push-off phase.

4.2.3 Joint moment and joint stiffness calculation (I, II, III, IV)

The jumping motions during either the DJ or sledge jumps (Max SSC and Max SJ) were recorded (100 frames·sec^{-1}) using a high speed video camera (NAC, HSV 200, Japan) from the right side of the subject. Body landmarks were attached as follows: head, wrist, elbow, shoulder, greater trochanter, knee, ankle and fifth metatarsal bone. The marker points of the body were digitized by a computerized digitizing system (APAS, Ariel Dynamics Inc., Calif., USA). The coordinates were filtered digitally through a

Butterworth type 4th-order zero-lag low-pass filter (cut-off frequency: 7 Hz) to remove noise. This was followed by calculation of the joint and segment angles and angular velocities as well as linear velocities. In addition, both the whole body's center of gravity (CG) and segment CG were calculated using the body segment parameters provided by Winter (1979). Net joint moments of the lower limb (hip, knee and ankle) were calculated by a basic link segment analysis (Brestler and Frankel, 1950) in conjunction with the kinematics and kinetic data. Joint extension power at each joint (hip, knee and ankle) was calculated by multiplying the joint moment with the joint angular velocity. In the present study, negative power refers to the eccentric action of the extensor muscles during the braking phase just after the touch-down. Correspondingly, positive power refers to the concentric action of the extensors in the push-off phase following the braking phase. The peak values of the negative and positive power of each jump were measured. The lower limb joints power curves during the Max SSC on the sledge are shown in Fig. 2.

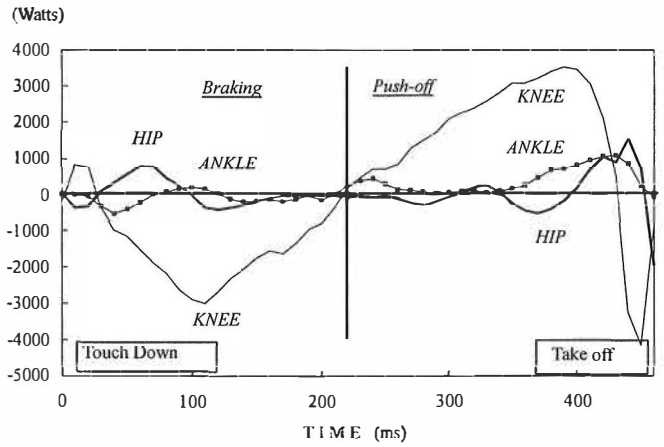

FIGURE 2 The lower limb joints power changes, calculated by a joint moment analysis, during the Max SSC in the same subject in figure 1. Note that the knee joint extensors are the main power generators during the Max SSC on the sledge.

The typical moment/angle relationship in the knee joint corresponding to the force/length relationship during DJ is presented in Fig. 3 B. This relationship was composed of three phases. The first phase shows the initial impact phase (P1) extending from the point of ground contact to the peak of the initial moment. The second phase (transmission phase (P2)) corresponds to the period from the initial peak to the onset of push-off. The third phase covers the concentric phase (P3). Stiffness was calculated by the coefficient of linear regression of the moment/angle relationship in each P1(initial stiffness) and P3 phase (concentric stiffness) according to previous reports (Allum and Mauritz, 1984, Blanpied and Smidt, 1992). Instantaneous stiffness was calculated as the instantaneous inclination of the joint moment – joint angle trajectory according to Dyhre-Poulsen and Laursen (1984) and Dyhre-Poulsen et al. (1991) using the following formula (I, III).

Stiffness $_{(n)}$ = (Mom $_{(n+1)}$ – Mom $_{(n-1)}$) / (Ang $_{(n+1)}$ – Ang $_{(n-1)}$)
where, Mom is the knee joint moment, Ang the knee joint angle, *n* the corresponding *n*th frame.
The first peak of instantaneous stiffness was analyzed to evaluate pre-landing stiffness (Fig. 3 C). A precise description of the instantaneous stiffness is given in original paper (I).

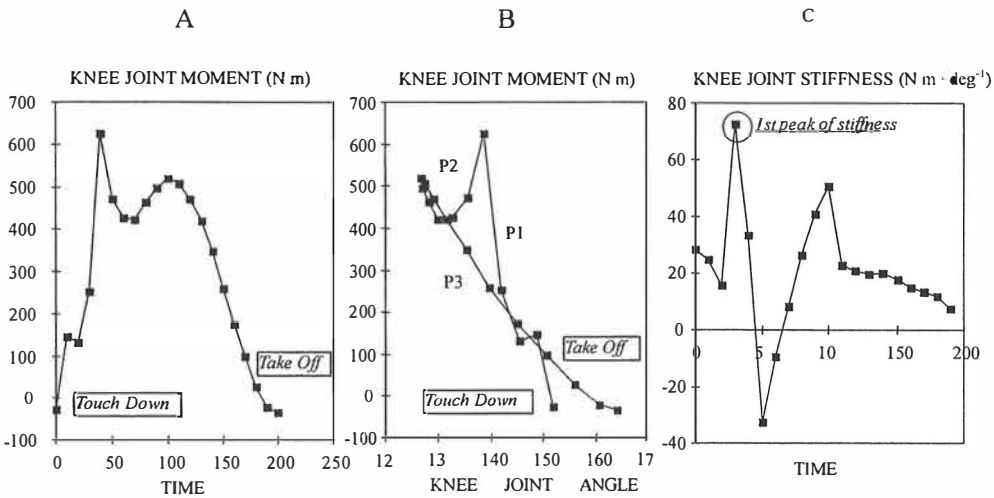

FIGURE 3 An example of the knee joint moment (A), knee joint moment – knee joint angle trajectory (B), and the knee joint instantaneous stiffness during drop jump (DJ) in one subject. Arrows in figure B indicate the direction of movement from touch down to take off in DJ.

4.2.4 Mechanical muscle model (III)

According to Crowe et al. (1980) and McMahon (1984), the knee extension moment generator can be expressed as a four-element linear mechanical muscle-tendon model including a series elastic component (SEC) with stiffness K_2, a parallel elastic element (PEC) with stiffness K_1, a damper (D) with viscosity B_1, and a contractile component (CC) with pure active tension T_1 (Fig. 4). In the present study, the rotational force (moment) was measured and therefore all the variables are expressed as a function of the knee joint angle. The basic equation for the moment at the knee joint (T) in this model can then be given as follows:

$$T = K_2\Delta\theta_2 = K_1\Delta\theta_1 + B_1\dot{\theta}_1 + T_1$$
$$\Delta\theta = \Delta\theta_1 + \Delta\theta_2$$

where θ is the length of the whole muscle tendon complex as a function of the knee joint angle, θ_1 the length of CC, and θ_2 the length of SEC. Each parameter (K_1, K_2, B_1, and T_1) is obtained by solving this equation in each phase (P1, P2, and P3) using the multiple regression technique (see Appendix). The knee extension moment represents the net muscle moment acting on the knee joint produced by the knee extensors and

flexors and by the biarticular gastrocnemius muscle. Therefore, each of the viscoelastic parameters of the model seems to contribute to the total sum of the value in each muscle around the knee joint. However, preferential activation of the knee extensors has been shown in DJ as judged by EMG activity (Voigt et al. 1995). Thus, it seems that the knee extensors are mainly responsible for this model. Although the triceps surae muscle is also active in DJ (Fukashiro et al. 1993), its role is not considered in the present analysis.

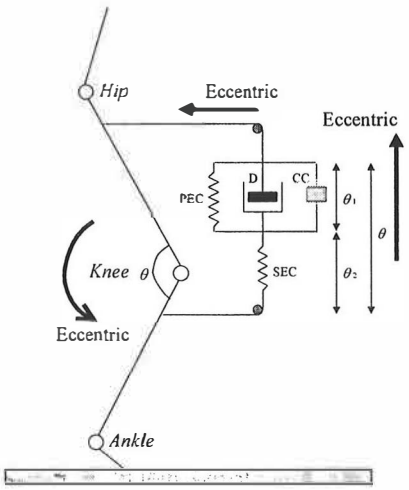

FIGURE 4 Mechanical model of the knee joint torque generator, based on Crowe et al., (1980) and McMahon (1984), and used in this study. SEC, PEC, D, and CC represent series elastic component, parallel elastic component, viscous damper, and contractile component, respectively.

It should be noted that this model does not distinguish between either tendon and muscle or subcutaneous tissue. In addition, individual muscle mass, training effect, and/or fiber composition could also affect each of the parameters. However, these factors are not taken into consideration in this model. Because of the insufficient time resolution of the kinematics (100 Hz), the limited number of data points in each time period (P1 – P3) could lead to error in the calculation of stiffness. Thus in the present study stiffness can be considered as estimated average stiffness during each time period.

4.2.5 Time – varying stiffness (VI)

In the present study, the lower leg was modeled as simple mass spring damper system, as described elsewhere (Crowninshield et al. 1976, Walshe et al. 1996). Equation of the motion of the system as a function of the time was as follows:

$$F(t) = M\ddot{x}(t) + c\dot{x}(t) + kx(t) \quad \text{---------- [1]}$$

where F is the reaction force, M the mass of the subject and the gliding part of the sledge, c leg viscosity, k leg stiffness, and x the displacement of the sledge sensed by the rotary encoder attached to the gliding part of the sledge. This model represents the total leg system as the combination of the three lower limb joints (hip, knee and ankle) and does not distinguish between the reflex component and intrinsic muscle component. Stiffness has been considered as controlled parameter basically regulated by the length feedback component originating from the spindle discharge and the force feedback component originating from the tendon organ discharge as well as the muscular component (Houk and Rymer 1981). Thus stiffness would be regulated instantaneously for load compensation in the natural circumstances such as the SSC action. Therefore, time-varying stiffness (TVS) was analyzed every 1 ms during the contact phase of the SSC action by using the time-varying moving correlation method which is a modification of the original method described by Bennett (1994). The precise description is as follows. In the equation 1, F, M, $\ddot{x}$, $\dot{x}$, x are known. Thus equation 1 can be rearranged as equation 2 as follows:

$$F(t) - M\ddot{x}(t) = c\dot{x}(t) + kx(t) \quad \text{---------- [2]}$$

When analyzing the corresponding time, (t)th data, a variance-covariance matrix was created by using the three sequential data sets of equation 2 (3 ms duration) consisting of the $(t\text{-}1)$th, (t)th and $(t+1)$th data to avoid phase shift of the estimated parameters. Within an interval the parameters c and k were assumed constant and then estimated with the conventional linear multiple correlation technique. These procedures were repeated every 1 ms during the contact phase of the SSC action during exercise. After the calculation, the average ensemble curves for stiffness and viscosity were constructed. A consideration of the significance of TVS in the SSC action has been fully described in the original paper (VI). Single trial TVS is shown in Fig. 5 accompanied by the corresponding ground reaction force and joint angle change. In the present study, the first peak, second peak and end of the braking phase of TVS were analyzed. In addition, the first peak as well as second peak of time-varying viscosity (TVV) were analyzed.

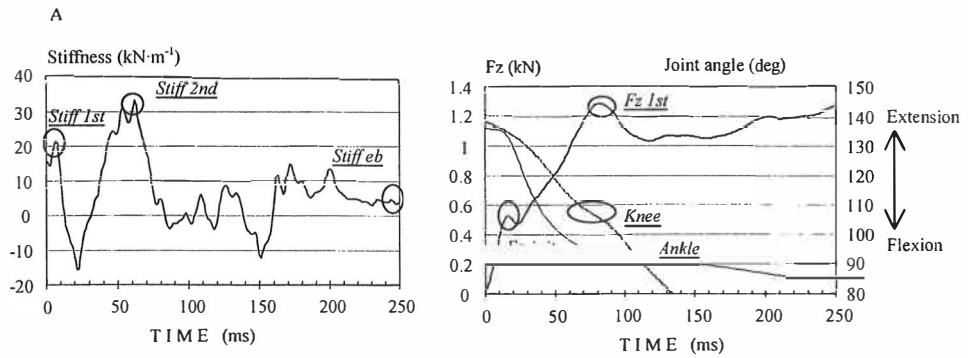

FIGURE 5 Sample record of the time varying leg stiffness (A) and kinetic and kinematic data (B) during the single SSC action in one subject. Small deflection point of the knee joint angle was denoted as eclipse. Some stiffness parameters also denoted as circle.

As discussed in the previous chapter, this TVS approach does not distinguish between the tendon and muscle or subcutaneous tissue. Individual muscle mass, training effect, and/or fiber composition also are not taken into account in this approach.

4.2.6 Measurement of Electromyography (EMG)

In the present study, surface EMG activity from the knee extensors (vastus lateralis (VL) and vastus medialis (VM)) as well as triceps surae muscle (medial gastrocnemius (GA) and soleus (SO)) of the right leg were recorded by silver chloride surface bipolar electrodes (Beckman miniature skin electrodes 650437, IL, USA) and transmitted telemetrically (Glonner, Germany). The electrodes were placed longitudinally over the muscle belly at an inter-electrode distance of 20 mm. Care was taken that the inter-electrode resistance was below 2 kΩ. The electrode positions were carefully marked on the skin to ensure the same electrode position in each test throughout the whole experimental period. The test-retest reproducibility of the EMG indicated by reliability coefficients has been reported to be relatively high: r = 0.94~0.97 for IEMG in jumping (Bosco 1982) and higher than 0.90 for most of the parameters in EMG patterns during SSC muscle actions (Gollhofer et al. 1990). The EMG signal was stored simultaneously with the force signal on a computer hard disk via a real time data acquisition system (Codas, Dataq Instruments Inc., OH, USA), which included a 12 bit A/D converter with a sampling frequency of 1 kHz. EMGs were integrated then time-normalized (aEMG) in the following three time periods; pre-activation before contact, braking phase (eccentric) and subsequent push-off phase (concentric). The pre-activation phase was defined as the 100 ms preceding ground contact (Komi et al. 1987a).

It is important to ensure that the EMG responses measured come only from the examined muscles. In the present study, cross-correlation analysis (Winter et al. 1994) was used to quantify possible cross-talk within adjacent muscle during the Max SSC performed by four subjects. The average cross-correlation coefficient (r_{xy}) between the VL and VM muscles was 0.10 ± 0.06 (P > 0.05). Furthermore, the GA and SO muscles also showed lower cross-correlation coefficient (r_{xy} = 0.18 ± 0.12, P > 0.05). Therefore, it was assumed that the extent of cross-talk was negligible in the present study.

4.2.7 Stretch reflex component in DJ (I)

The best performance of 3 trials in each jump was used in analyzing EMG, kinematics and stiffness. The EMG signal was full-wave rectified at 500 Hz. To analyze the stretch reflex response during jumping, the rectified EMG signal of the best trial was low-pass filtered at 75 Hz (butterworth type 4th order zero lag digital filter). Then the short latency M1 and medium latency M2 components of the stretch reflex were identified according to the original definition of Lee and Tatton (1978). The pre-activation phase was also defined as 100 ms preceding ground contact (Komi et al. 1987a). In the present study, VL muscle has been assumed as the main knee extensor muscle to examine the stiffness and reflex interaction of the knee joint. Thus the reflex component of the VL muscle was identified. Fig. 6 A shows the identification of the pre-activation, M1 and M2 responses during a single DJ trial and a comparison between the raw and filtered EMG signal of the VL muscle in one subject. Usually, a rapid EMG burst appeared with approximately 30 ms latency after ground contact followed by moderate EMG wave having about 60 ms latency. This rapid EMG burst corresponded to a short latency stretch reflex M1, as suggested by Dietz et al. (1979 1981). Similarly, the second response with a medium latency was a

labeled as M2. These latencies were in good agreement with the value of Bergui et al. (1992), who reported 32.2 ms of M1 latency and 57 ms of M2 latency in the stretch reflex of the quadriceps femoris muscle. In addition, these latencies of the knee extensor muscles were quite similar to the original values of Lee and Tatton (1978), who reported 30~35 ms latency of M1 and 55~65 ms latency of M2 in the arm muscles. These values seem therefore acceptable when considering the similar distance to the reflex center as suggested by Bergui et al. (1992). Lee and Tatton have also identified M3 components with a latency of 90~100 ms. However, it is possible that voluntary activation is also included in this component (Fellows et al. 1993). Therefore, the analysis of EMG parameters included areas of M1, M2 and the pre-activation of EMG waves above the zero level.

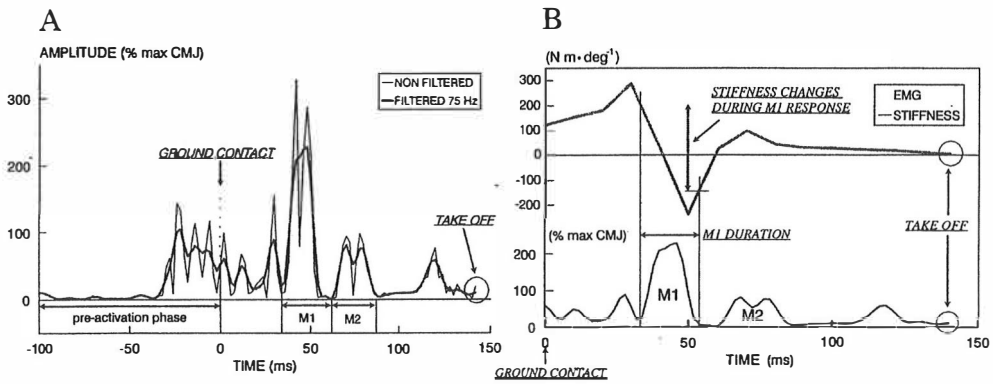

FIGURE 6 Left hand side (A) shows nonfiltered raw and low pass filtered (75 Hz) full-wave rectified EMG of VL muscle during single DJ trial. Thin line shows nonfiltered EMG wave. Thick line indicates filtered EMG signal. Pre-activation phase, M1 and M2 components are also indicated by the arrows. Right hand side (B) shows the calculation of stiffness changes during M1 response in the single DJ trial.

In the Fig. 6 B (*upper trace*), stiffness shows a rapid decline after the 1st peak, which appeared 20 ~ 30 ms after contact. This period corresponds to the intervention of the short latency reflex (Fig. 6 B, *lower trace*). Thus it is possible that the amount of stiffness changes from a higher positive value to a lower negative value during the short latency M1 response characterizing the changes in reflex activity. A phase relationship between stiffness and the short latency stretch reflex has been suggested earlier (Allum and Mauritz 1984, Houk et al. 1981). The change in stiffness during the short latency reflex activity was calculated to assess the relationship between stiffness and the short latency stretch reflex (Fig. 6 B, *vertical arrow*). The possible phase relationship between stiffness and short latency stretch reflex has been fully described in the original paper (I). In the present study, all the calculations of the mechanical parameters, including joint moment and stiffness as well as EMG filtering, were done by the special computer program written by BASIC (Quick BASIC ver 4.0, Microsoft, USA).

4.2.8 Blood analysis (I, II, IV, V, VI)

Blood lactate (LA) concentration was determined at rest, immediately before and after the sledge exercise as well as 3, 5 and 25 min later (end of the session), using a lactate

analyzer (model 640, Roche). Serum creatine kinase (CK) activity was measured at rest, immediately before and after the sledge exercise as well as before every subsequent testing session. CK activity was assayed using a CK ultra violet test kit (Boehringer Mannheim, Mannheim, F. R. G.).

4.2.9 Statistics

All values are presented as means ± SD. Pre-post fatigue changes were tested by analysis of variance (ANOVA) for repeated measurements on one factor and then post hoc LSD multiple comparison was performed. The level of significance was set at $P<.05$. Thereafter, all values were converted into relative changes (Δ %) from the pre-fatigue level for the correlation analysis. With reference to the succession of an initial and a delayed secondary phase of injury following muscle damage (Faulkner *et al.* 1993, MacIntyre *et al.* 1996), it is possible that the secondary injury effect could be contaminated by the initial injury effect. Therefore, the effects of fatigue on the parameters measured were expressed as relative changes as compared to the preceding test values using the following formula (II):

$$\text{(n)th inter test relative change} = \frac{\text{(n - 1)th test value}}{\text{(n)th test value}} \times 100$$

where n number of test session, e.g before = 1, after = 2,.....4th day = 5

The correlation coefficient was used to compare the changes in different parameters between sessions. All statistical calculations were performed with the statistical package (STATISTICA ver. 4.5, StatSoft, USA).

5 RESULTS

The most important findings obtained from the present series of experiments are presented below. For more details the original papers (I – VI) should be consulted.

5.1 Interaction between pre-landing activities and SSC performance

The relationships between DJ performance (take off velocity) and individual joint mechanical variables (initial stiffness P_1, concentric stiffness P_3 and positive peak power) are shown in Table 4. DJ performance correlated positively with the knee joint moment at the end of stretch ($P < 0.01$) with knee joint concentric stiffness ($P < 0.01$) and with knee joint positive peak power ($P < 0.01$). In addition, these stiffness and power variables were significantly intercorrelated. However, the take-off velocity correlated neither with ankle joint positive peak power nor with concentric stiffness. Significant correlations were found at the knee joint between SEC stiffness in the P2 phase of the knee joint and between knee joint concentric stiffness ($r = 0.80$, $P < 0.01$) and the 1st peak of knee joint instantaneous stiffness ($r = 0.88$, $P < 0.01$). Interactions between these stiffness variables are demonstrated in Fig. 7. Multiple regression analysis showed that SEC stiffness in the P2 phase of the knee joint can be explained by a combination of the pre-activity of VL muscle and knee joint angular velocity at touch-down ($F = 5.76$, $P < 0.05$). DJ performance was fitted by the second order polynomial function of the average pre-landing angular velocity of the knee joint during 50 ms before touch-down when the knee joint is being flexed momentarily (Fig. 8). DJ performance can also be explained by a combination of the pre-landing angular velocity of the knee joint and SEC stiffness in the P2 phase of knee the joint ($F = 15.95$, $P < 0.01$) (Table 5). With respect to these results, Fig. 9 shows the proposed two types of jump motion with regard to the pre-landing motion of the knee joint.

TABLE 4 Correlation matrix between performance in DJ and the mechanical variables in the knee joint and the ankle joint.

		1	2	3	4	5	6	7
Take off velocity	1							
Ground contact time	2	-0.62						
Knee initial stiffness P1	3	0.42	-0.59					
Knee concentric stiffness P3	4	0.79 **	-0.58	0.43				
Knee moment at the end of stretch	5	0.84 **	-0.35	0.40	0.84 **			
Knee joint positive peak power	6	0.82 **	-0.56	0.37	0.72 *	0.86 **		
Ankle joint positive peak power	7	0.10	-0.24	-0.14	0.37	0.26	0.27	
Ankle concentric stiffness P3	8	0.06	-0.13	0.07	0.54	0.43	0.39	0.74 *

*, ** Significant at $P < 0.05$ (*), $P < 0.01$ (**) (n=9)

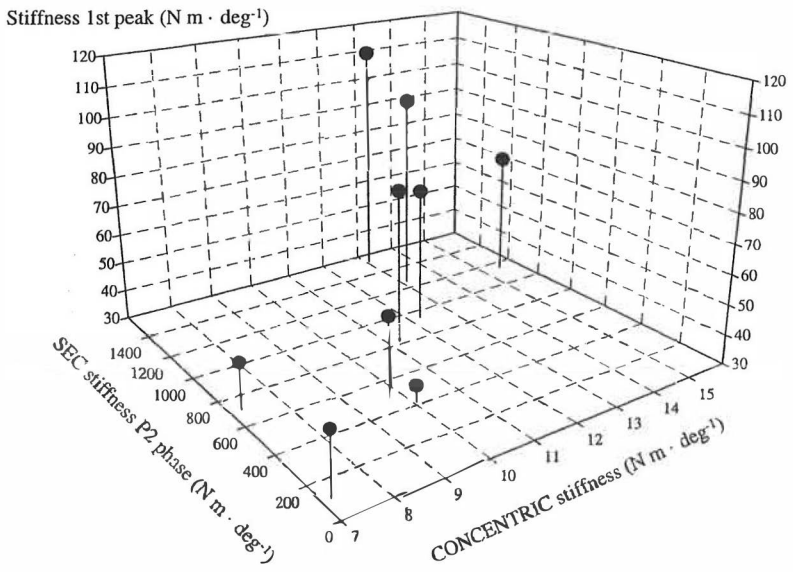

FIGURE 7 Interactions between the 1st peak of stiffness, SEC stiffness during P2 phase and the concentric stiffness in the knee joint during DJ.

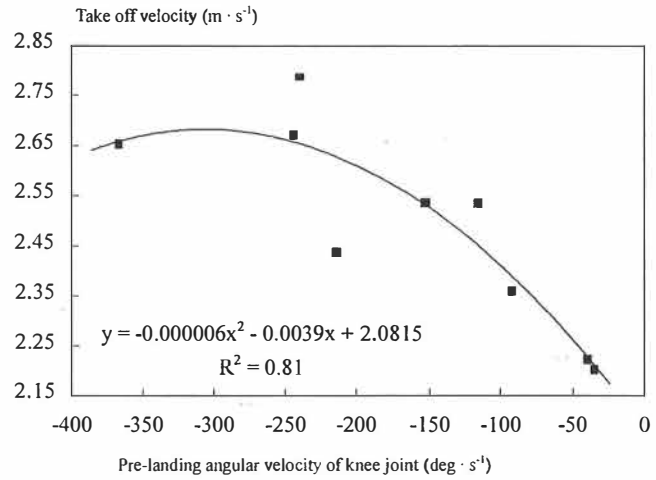

FIGURE 8 Relationship between the take off velocity in DJ and the average pre-landing knee joint angular velocity during 50 ms preceding the touch down in DJ. The negative angular velocity corresponds to the flexion movement of the knee joint.

TABLE 5 Multiple correlation table in DJ

Dependent variables	Independent variables	R	R^2 adjusted	F	Standard Error
SEC stiffness P2 (standard partial reg. coeff.)	69.28 × VL pre-activity – 3.39 × Knee joint angular velocity at touch down – 434.36 (0.92*) (-0.82*)	0.81	0.54	5.76 *	303
Take off velocity (standard partial reg. coeff.)	-0.001155 × Pre-landing angular velocity of knee joint + 0.000205 × SEC stiffness P2 (-0.62**) (0.46*) + 2.1292	0.92	0.79	15.95 **	0.09
Take off velocity (standard partial reg. coeff.)	-0.004154 × Contact time + 0.000014 × Max RFD + 3.0767 (-0.67*) (0.55)	0.83	0.59	6.73*	0.13

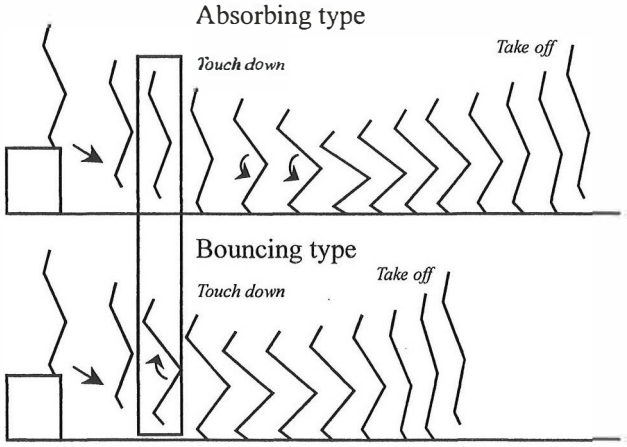

FIGURE 9 Proposed jump motion based on the result from the present study.

5.2 Basic physiological responses after exhaustive SSC exercise

In the fatigue experiment (exp. 2 – 4), the same protocol was used to induce exhaustive SSC fatigue. The general physiological responses are summarized in Table 6. In each experiment, the subjects repeated the rebound jumps around 100 times, which took about 3 min and were accompanied by increasing contact time. A relatively high lactate value indicates the occurrence of metabolic fatigue. In addition, serum CK activity increased 2 days after the exercise. Therefore the present SSC loading seems to induce both metabolic fatigue and possible delayed muscle damage, as suggested earlier (Nicol et al. 1996).

TABLE 6 General physiological responses after the exhaustive SSC exercise.

		Experiment 2	Experiment 3	Experiment 4
Number of repetition (times)		92 ± 30	117 ± 70	87 ± 24
Exercise duration (min)		2.7 ± 0.8	2.9 ± 1.7	2.5 ± 0.7
Contact time (ms)	Initial	527 ± 83	—	554 ± 90
	Last	692 ± 95***		702 ± 100***
Blood Lactate ($mmol \cdot l^{-1}$)	Before	1.6 ± 0.3	2.4 ± 0.7	1.5 ± 0.3
	After	7.2 ± 0.8***	12.5 ± 2.6***	7.1 ± 0.8***
Serum CK ($U \cdot l^{-1}$)	Before	214 ± 38	166 ± 188	211 ± 41
	2 days after	486 ± 300***	540 ± 407***	477 ± 272***

*** Significant at $p < 0.001$

5.3 SSC fatigue effect on the pure concentric action

The changes in jumping performance (jump height) in the Max SSC and Max SJ are shown in Fig. 10. The Max SJ performance decreased immediately after the SSC exercise ($P < 0.0001$). However it recovered quickly within 10 min and 20 min after exercise, and remained at the same level during the subsequent follow up session (2 days and 4 days after exercise). On the other hand, the Max SSC performance showed a delayed decrease on day 2 after exercise ($P < 0.01$). Thus these two jumps demonstrated different patterns of change in performance during the follow-up period of 4 days.

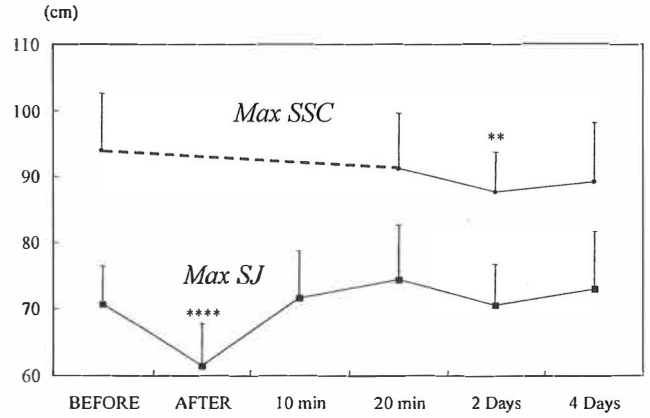

FIGURE 10 Changes in the jumping performance in the Max SJ and Max SSC during the four day follow-up period.

Changes in EMG activity are shown in Fig. 11 and 12 in which all the EMG values are expressed as a percentage of the before-fatigue values. Absolute EMG values (microvolts) were used for the calculation of the significant difference from the before-fatigue level. In the Max SJ, the EMG activity showed no significant changes after the SSC exercise (Fig. 11). GA EMG showed on the average, a 20 % decrease immediately after fatigue. However, due to the large inter-individual variations this change was statistically non significant. In the Max SSC, the pre-activity of the knee extensors (VL and VM) decreased on day 2 ($P < 0.01$) and day 4 ($P < 0.01$) after exercise (Fig. 12). The GA muscle also showed a delayed decrease day 4 after the exercise ($P < 0.05$). During the braking phase, in which the extensor muscles perform eccentric action, the EMG activity of the triceps surae muscle (SO and GA) and VM muscle decreased on day 2 and day 4 after exercise ($P < 0.01 - 0.001$). In the push-off phase, in which the extensor muscles perform concentric action, GA muscle EMG activity showed a continuous decline from 20 min after to the subsequent follow up periods (2 days and 4 days after exercise, $P < 0.01 - 0.001$).

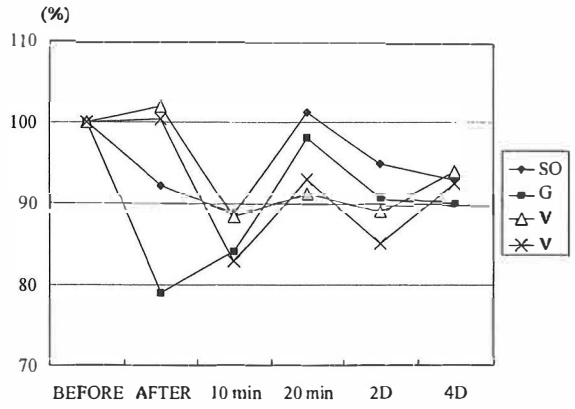

FIGURE 11 Relative changes in the EMG activities of the Max SJ during the fourth day follow-up period. The values are expressed as relative from the before fatigue measurements.

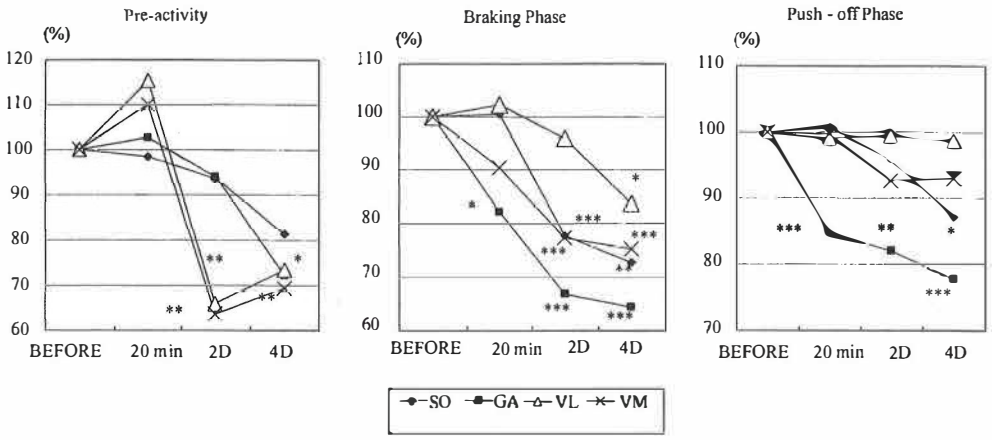

FIGURE 12 Relative changes in the EMG activities during pre-activation phase (left), braking-phase (middle) and push-off phase (right) of the Max SSC during the follow-up period after the SSC exercise.

5.4 Effect of exhaustive SSC exercise on reflex and stiffness interaction in DJ

DJ performance (flight time) decreased significantly immediately after (492 ± 33 ms), 2 hours after (494 ± 35 ms) and 2 days after (493 ± 32 ms, $P < 0.01$) the SSC fatigue exercise as compared to before (511 ± 40 ms). Ground contact time increased significantly 2 hours after (229 ± 44 ms, $P < 0.01$), 2 days after (224 ± 38 ms, $P < 0.05$) and 4 days after (223 ± 45 ms, $P < 0.05$) the SSC fatigue exercise as compared to before exercise (205 ± 38 ms). Knee extension positive peak power in the DJ decreased

significantly immediately after (1915 ± 643 W, $P < 0.05$), 2 hours after (1804 ± 562 W, $P < 0.01$) and 4 days after (1872 ± 742 W, $P < 0.01$) the SSC fatigue as compared to pre-fatigue test (2364 ± 564 W). There was a decreasing trend in knee extension positive peak power in all the test conditions. Figure 13 shows the averaged EMG wave (n=10) during DJ before and immediately after the SSC exercise. It can be seen that increased pre-activation, a decreased M1 component and an increased M2 component are apparent immediately after fatigue.

Mean group changes in pre-activation, in M1 and M2 stretch reflex latencies, duration and area are shown in table 7. Pre-activation before contact increased immediately after ($P < 0.01$) and 2 hours after ($P < 0.01$) the SSC fatigue. The M1 area decreased continuously throughout the test period, although not significantly. In contrast, the M2 area increased 2 days after ($P < 0.05$) the SSC exercise. The latency and duration of both the M1 and M2 components did not show any significant changes except for M2 latency 2 days after measurement. Both the 1st peak of the stiffness and the stiffness changes during the VL M1 response decreased significantly 2 hours and 2 days after SSC fatigue (table 7). The immediate post exercise change in the knee positive peak power was positively correlated ($P < 0.05$) with the respective change in the VL stretch reflex M1 area during the DJ (figure 14, *upper left*) and negatively ($P < 0.01$) with respective to the change in ground contact time (figure 14, *upper right*). Change in stiffness during the VL M1 response immediately after the SSC exercise was positively correlated ($P < 0.05$) with the respective change in VL stretch reflex M1 area (figure 14, *lower left*) and negatively ($P < 0.01$) with respective change in ground contact time (figure 14, *lower right*). In addition, the delayed increase of CK on the 2nd and 4th day post exercise was negatively correlated ($P < 0.05$ and $P < 0.01$) with the changes in DJ performance.

TABLE 7 Changes in pre-activity, stretch reflex parameters, and stiffness parameters.

	Before	After	2 h after	2 days after	4 days after
Pre-activity @	12.9±6.9	18.4±8.7**	19.1±6.3**	13.9±8.0	13.5±6.9
M1 Latency (ms)	29.8±6.3	30.2±3.8	27.4±4.9	29.2±5.9	31.4±6.7
M1 Duration (ms)	28.2±4.8	25.8±4.6	26.8±4.2	24.0±6.2	24.2±5.1
M1 Area @	1.7±0.8	1.2±0.7	1.4±0.8	1.4±1.1	1.1±0.7
M2 Latency (ms)	58.0±6.0	56.0±5.7	54.2±3.7	53.2±3.9*	55.6±8.1
M2 Duration (ms)	24.0±5.3	27.0±6.3	25.2±6.8	28.2±5.0	27.6±7.0
M2 Area @	1.0±0.5	1.4±0.7	1.5±0.8	1.6±1.1*	1.5±0.7
1st peak of stiffness (N m·deg^{-1})	91 ± 73	68 ± 24	64 ± 25*	63 ± 41*	75 ± 59
stiffness changes in VL M1 (N m·deg^{-1})	108 ± 68	70 ± 46	57 ± 40**	65 ± 51*	74 ± 74

@ Expressed as arbitrary unit (n=10)

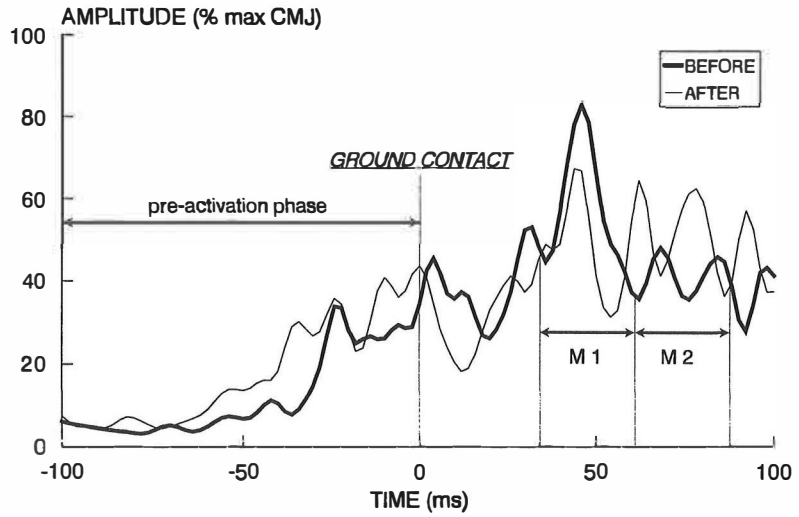

FIGURE 13 Average (n=10) filtered EMG wave during DJ before and immediately after the SSC exercise. Note the increased pre-activity, decreased M1 response and increased M2 response.

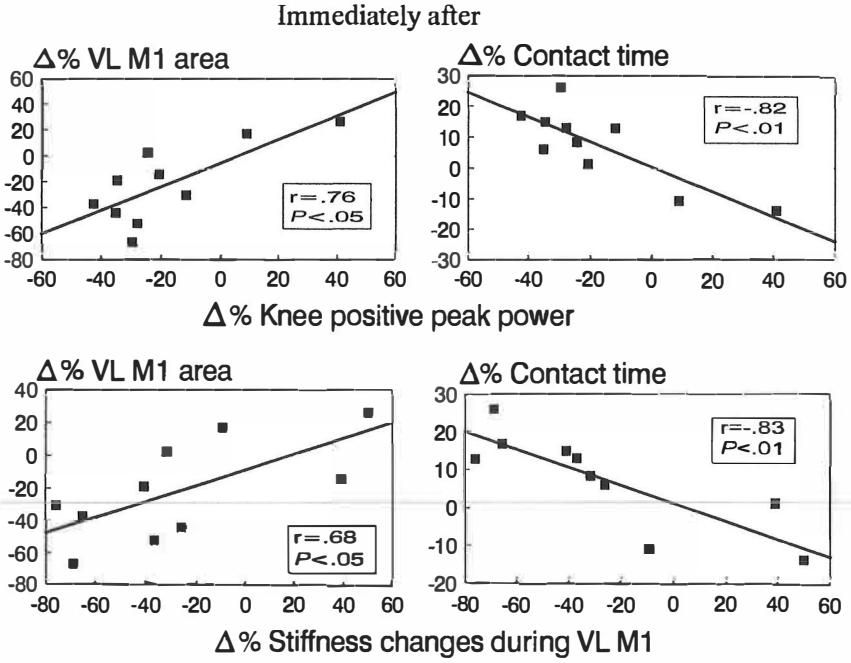

FIGURE 14 Correlation between changes in the knee extension positive peak power of DJ immediately after the SSC exercise and corresponding changes in VL M1 area (upper left) and ground contact time in DJ (upper right). Lower column show the correlation between changes in stiffness during VL M1 response of DJ immediately after the SSC exercise and corresponding changes in VL M1 area (*lower left*) and ground contact time in DJ (*lower right*).

5.5 Effect of exhaustive SSC exercise on the time course of mechanical behavior in the DJ

The relative changes in DJ performance between before and immediately after the exhaustive SSC exercise (BEF - AFT) was negatively related to the relative changes in DJ performance between immediately after and 2 hours after exercise (AFT - 2H) ($r = -0.73$ $P < 0.05$). This relationship indicates that the decreased performance immediately after the exercise corresponds to the increased performance 2h after. This would further indicate that the subject who presented the largest decrease in performance immediately after exercise had recovered well 2 h later. In addition, the relative changes in the knee joint moment at the end of stretch between AFT - 2H was negatively related to the relative changes in DJ performance between 2 hours after and the 2nd day after the exercise (2H - 2D) ($r = -0.81$ $P < 0.01$). Thus the early recovery 2h after exercise was related to the decrease in performance 2 days after exercise.

The relative changes in the knee joint moment at the end of stretch between BEF - AFT were negatively related to the initial CK changes between AFT - 2H ($P < 0.05$, Fig. 15 A). The subsequent changes in the knee joint moment at the end of this phase between AFT - 2H were further related to the secondary increase in CK between 2H - 2D ($P < 0.01$, Fig. 15 B). Therefore the acute fatigue effect immediately after the SSC exercise was related to the initial increase in CK activity 2h after. In addition, the early recovery 2h after related to the secondary increase in CK 2 days after exercise.

The relative changes in initial stiffness between 2H - 2D were related positively to the corresponding changes in DJ performance ($P < 0.001$, Fig. 16 A) and negatively to the corresponding changes in the knee joint angle at touch down during the DJ ($P < 0.001$, Fig. 16 B). Thus the additional decrease in performance 2 days after exercise would be accounted for by the corresponding decrease in initial stiffness, which was associated with the increase in knee extension at touch-down. A jump motion can therefore be proposed regarding the altered kinematics as well as stiffness (Fig. 17). Relative changes in CK activity, VL pre-activity and DJ performance showed a contrasting pattern between a delayed increase in CK activity and a decrease in VL pre-activity on day 2 after the SSC exercise (Fig. 18).

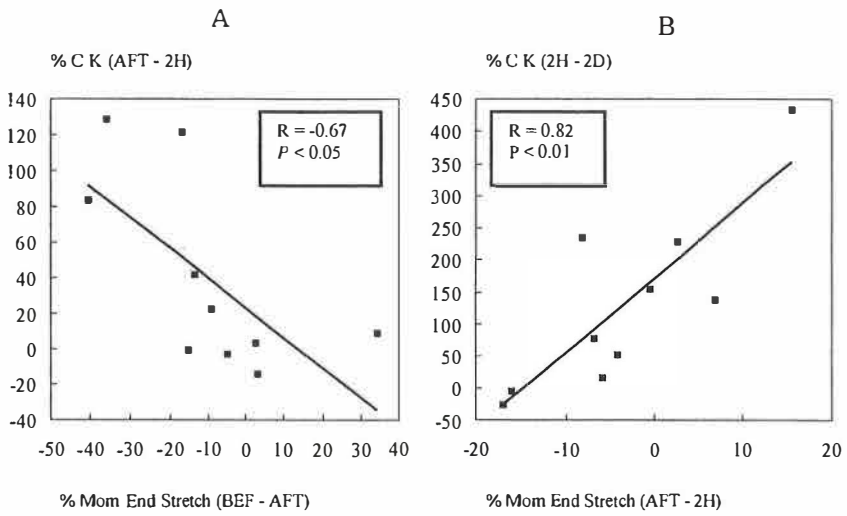

FIGURE 15 A: Relationship between relative changes in the Mom End Stretch between BEF - AFT and relative changes in the CK between AFT - 2H. B: Relationship between relative changes in the Mom End Stretch between AFT - 2H and relative changes in the CK between 2H - 2D.

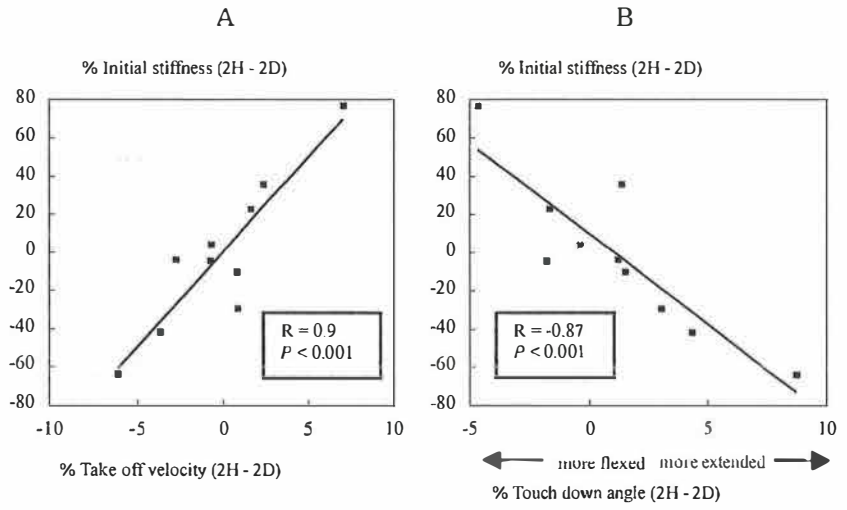

FIGURE 16 A: Relationship between relative changes in the initial stiffness of the knee joint between 2H - 2D and relative changes in the DJ performance between 2H - 2D. B: Relationship between relative changes in the initial stiffness of the knee joint between 2H - 2D and relative changes in the knee joint angle at the touch down in DJ between 2H - 2D.

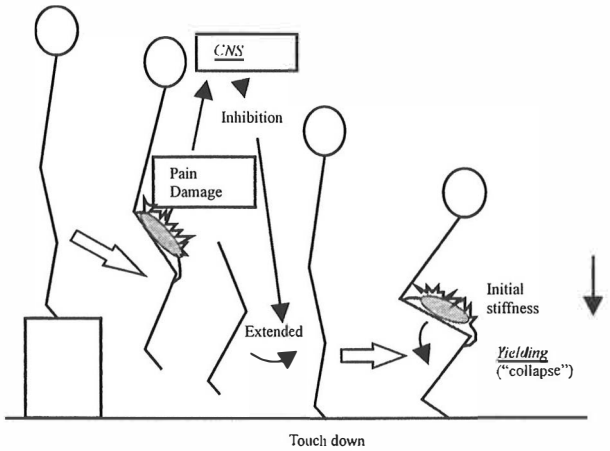

FIGURE 17 Proposed interaction between pre-landing motor command and stiffness regulation at touch down in the drop jump after the occurrence of muscle damage. CNS: central nervous system.

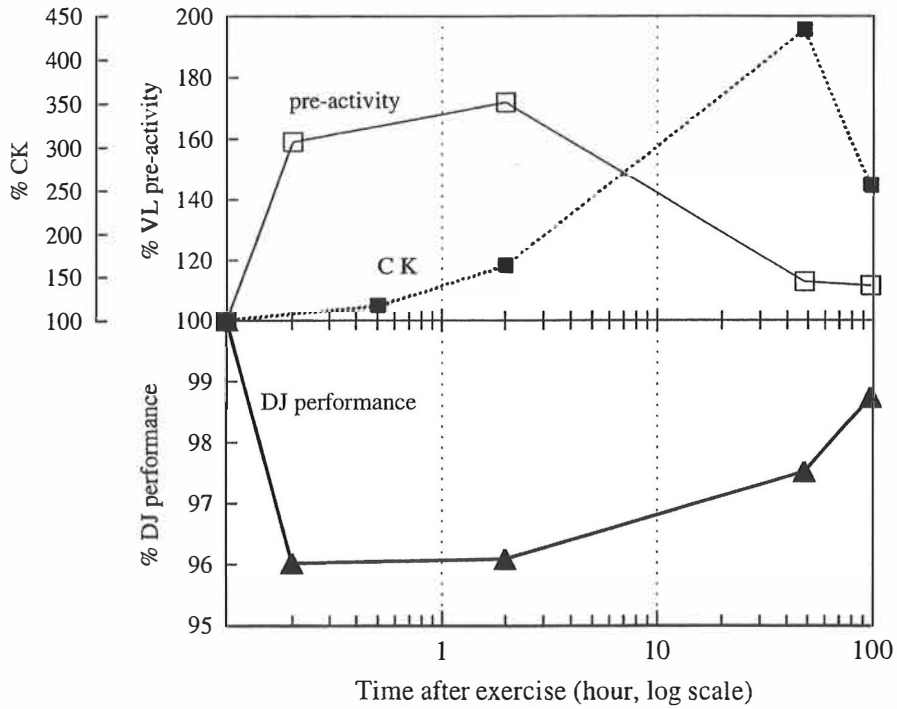

FIGURE 18 Percentage changes in creatine kinase (CK) activity, pre-activity of the VL muscle and DJ performance after the exhaustive SSC exercise.

5.6 Adaptation process of the neuromuscular system during exhaustive SSC exercise

The average curves of the ground reaction force, ankle and knee joint angle during the SSC exercise are shown in Fig. 19 with the normalized time axis. The contact time in each rebound jump progressively increased from the initial stage (554 ± 90 ms) to the last stage (702 ± 100 ms, $P < 0.001$) of the exercise. As shown in figure 19, the average force curve clearly indicates the depression in force output during the last stage of the exercise as compared to the initial stage. Therefore the decreased force output was compensated by increased contact time to achieve the same external work output. The joint kinematics of the ankle and knee joints were modified during the exhaustive SSC exercise. These modifications were seen in both the flight and contact periods of the SSC exercise. The average TVS and TVV curves during the braking phase of the sledge exercise are shown in Fig. 20. In the TVS, the first peak (Stiff 1st), second peak (Stiff 2nd) and end of the braking phase (Stiff *eb*) decreased along with the time course of the SSC exercise. In contrast, the second peak of the TVV (Vis 2nd) gradually increased at the last stage of the SSC exercise.

Relative changes in kinetics and kinematics during the time course of the SSC exercise are shown in Fig. 21. Both the ankle joint flexion ROM (range of motion) and the knee joint extension ROM during the flight period increased linearly immediately after the initial stage of the SSC exercise. During the contact period, the change in Knee ROM showed a bimodal pattern that consisted of an initial small increase and subsequent plateau until the middle stage of the exercise (60 % of the total number of jumps) followed by a secondary large increase during the last stage of the exercise. The ankle ROM also showed a similar pattern to that observed in the knee joint but in the opposite direction. Therefore a symmetrical relationship was found between Knee ROM and Ankle ROM during the contact phase of the rebound jump throughout the time course of the fatiguing exercise. Changes in contact time and Fz *eb* (force at the end of braking phase) demonstrated a similar symmetrical pattern as observed in Knee ROM and Ankle ROM during the contact phase. To summarize, repeated exhaustive SSC exercise induced alterations in kinematics and kinetics during the SSC exercise. This was especially clear in the kinematics of the flight phase in which approximately 10 - 20 times greater modifications were found as compared to the contact period. Relative changes in aEMG activity are shown in Fig. 22. Contrasting aEMG activity was seen between the knee extensors (increasing) and ankle extensors (decreasing) during the contact period throughout the time course of the fatiguing SSC exercise. In addition, changes in the activity of the knee extensors during the braking phase showed a similar turning point (60 % of the total number of jumps) so that observed in the changes in joint kinematics (Knee ROM and Ankle ROM), contact time and kinetics (Fz *eb*) during the contact period.

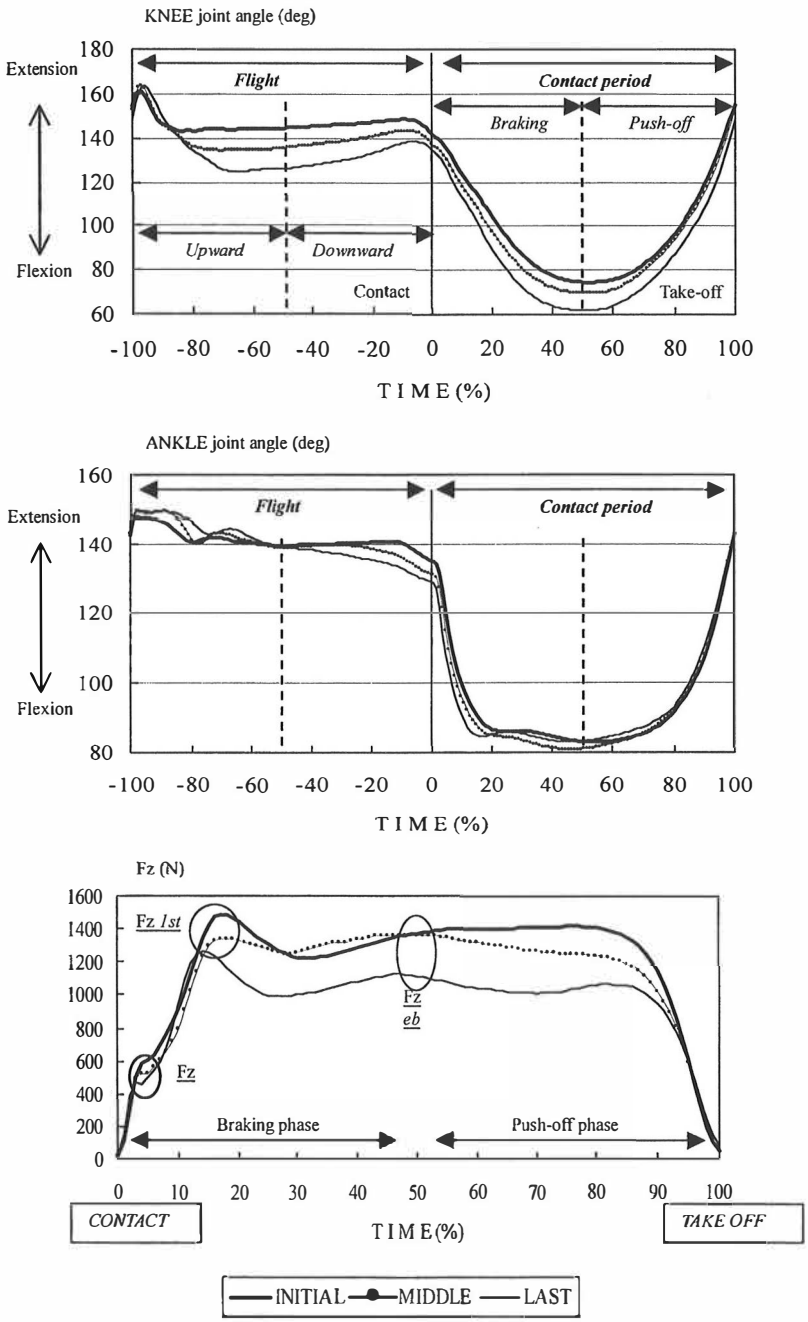

FIGURE 19 Average curves (n=80) of the knee joint angle (top), ankle joint angle (middle) and ground reaction force (bottom) in each stage of the stretch-shortening cycle (SSC) exercise (bold line, initial stage; thin line with circle dot, middle stage; thin line, last stage). Contact period normalized as 100 %. Flight period normalized as –100 %. In the reaction force, initial shoulder (Fz init), first peak (Fz 1st) and force level at the end of braking phase (Fz eb) are denoted as circle.

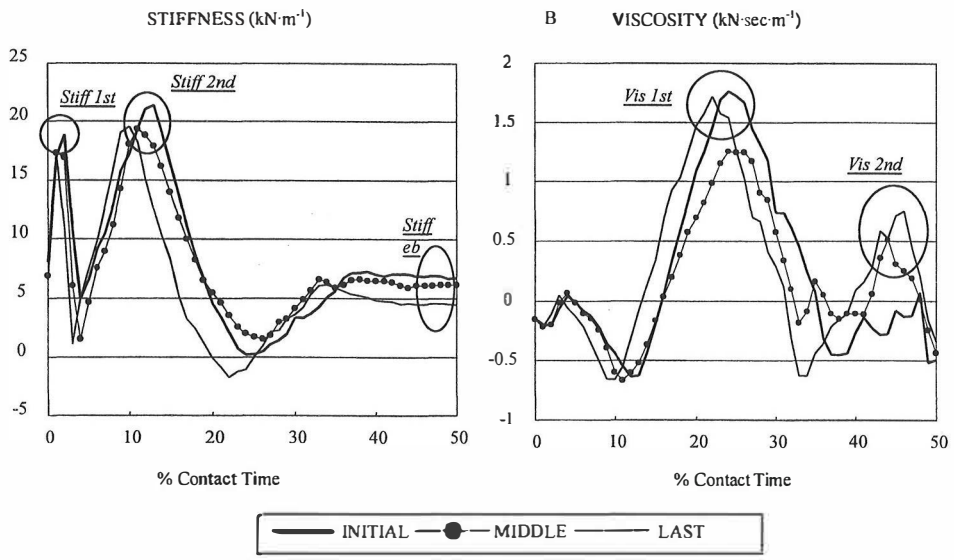

FIGURE 20 Average curves (n=80) of the time-varying leg stiffness (A) and time-varying leg viscosity (B) during braking phase of the SSC exercise in each stage of the exercise. Time axis was normalized as same as figure 19. Stiffness and viscous parameters are also denoted as circle.

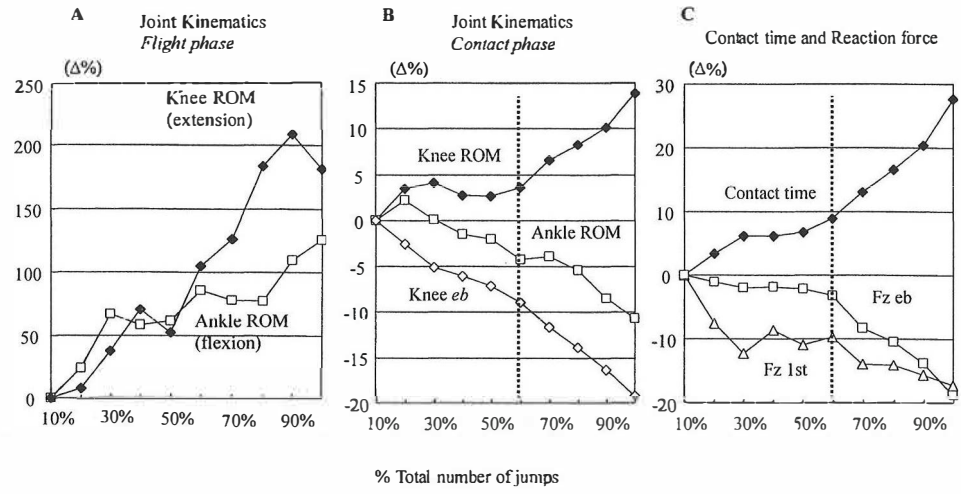

FIGURE 21 Relative changes in kinetics and kinematics during time course of the SSC exercise. A: joint kinematics during flight phase. B: joint kinematics during contact phase. C: contact time and reaction force. ROM; range of motion. Knee eb: knee joint angle at the end of braking phase. Fz eb: force level at the end of braking phase. Fz 1st: first peak of reaction force. Horizontal axis indicates the total number of jumps as relative value. 10 %: initial stage of the exercise. 100 %: last stage of exercise.

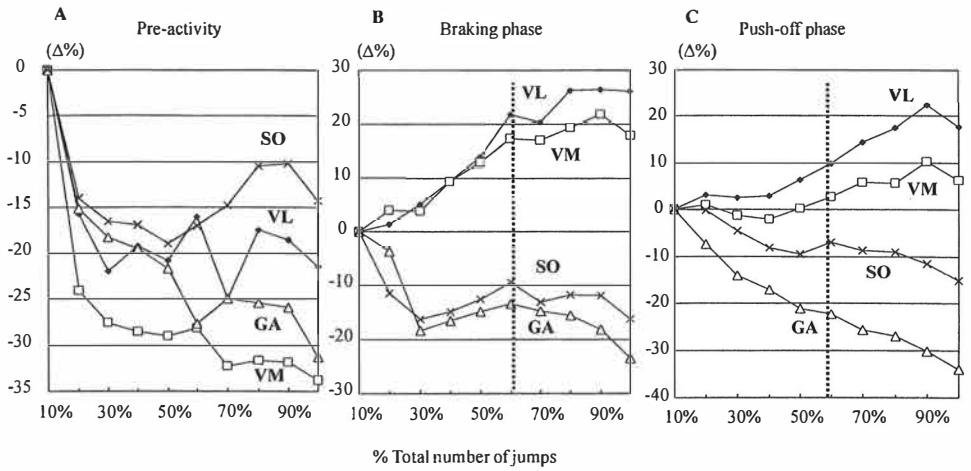

FIGURE 22 Relative changes in EMG activities of the VL, vastaus medialis (VM), GA and soleus (SO) muscles during time course of the SSC exercise. A: pre-activity. B: braking phase. C: push-off phase.

As shown in Figs. 21 and 22, the turning point of the fatigue response could be seen after the middle stage of the exercise (60 % of the total number of jumps) as observed in joint kinematics (Knee ROM and Ankle ROM), contact time and kinetics (Fz *eb*) during the contact period of the rebound jump. At this stage, the decrease in pre-activity of the VL muscle and increase in ankle flexion velocity during the downward period were related to the increase in Knee ROM (Fig. 23 A, B). The increase in Knee ROM also was related to the increase in contact time (Fig. 23, C). In addition, the relative decrease in Stiff 1st was related to the relative increase in the pre-landing ankle flexion velocity ($P < 0.05$), contact time ($P < 0.01$) and viscous ratio ($P < 0.05$) as well as to decrease in Stiff *eb* ($P < 0.05$) at the second half of the exercise (70 % of the total number of jumps, Fig. 24). The increased viscous ratio during the second half of the exercise (70 % of the total number of jumps) was further related to the decrease in Knee *eb* (knee joint angle at the end of the braking phase, $r = -0.81$, $P < 0.05$) and the delayed increase in CK activity 4 days after exercise ($r = 0.83$, $P < 0.01$).

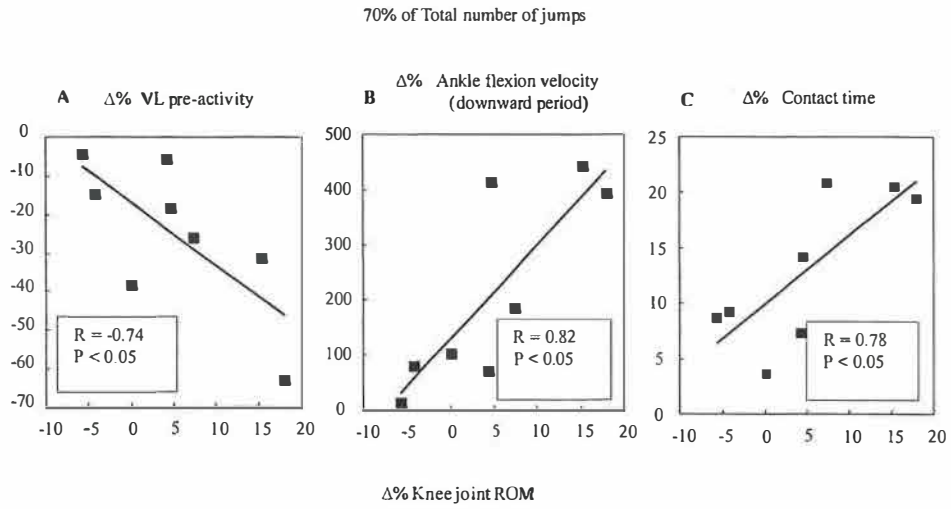

FIGURE 23 Correlation between changes (Δ) in range of motion of the knee joint and VL pre-activity (A), ankle flexion velocity during downward period (B) and contact time (C). All values were collected at second half of the SSC exercise (70 % of total number of jumps).

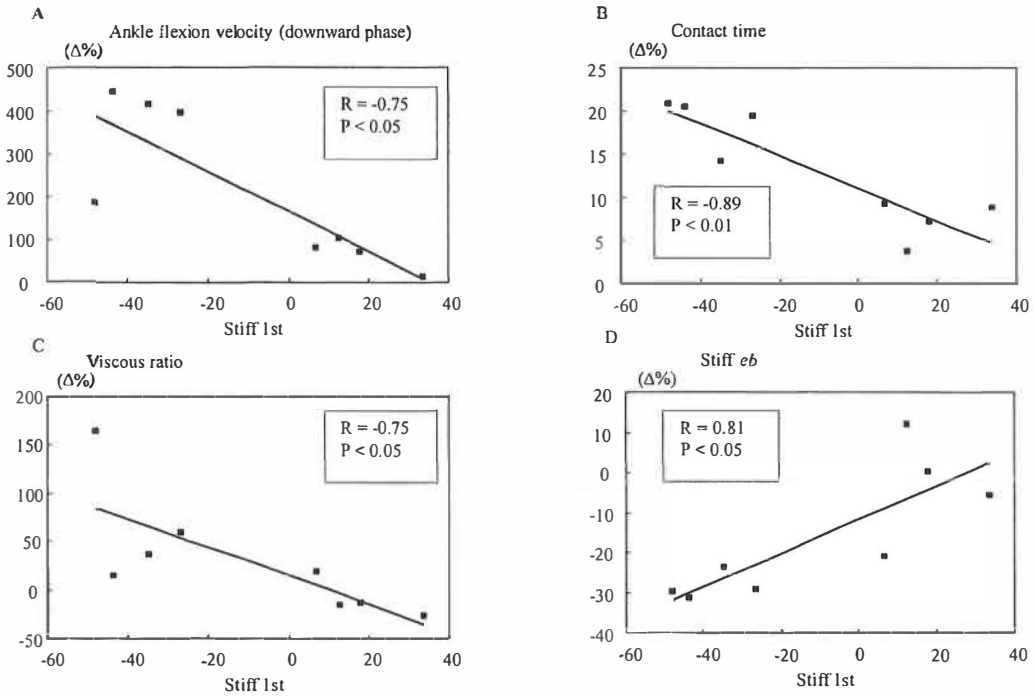

FIGURE 24 Correlation between changes (Δ) in Stiff 1st and ankle flexion velocity during downward phase (A), contact time (B), viscous ratio (C) and Stiff eb (D). All values were collected at second half of the SSC exercise (70 % of total number of jumps).

6 DISCUSSION

The main findings in the present study were as follows:

(1) In the non fatigue condition, SSC (DJ) performance was related to knee joint positive peak power and concentric stiffness. The more comprehensive approach using the mechanical muscle model revealed the sequential linkage of the stiffness regulation mechanism from the initial contact period to the concentric phase via higher SEC stiffness. Multiple regression analysis showed that DJ performance could be explained by the combination of SEC stiffness and pre-landing knee joint angular velocity.

(2) Exhaustive SSC exercise induced an acute performance deficit on the pure concentric action immediately after exercise. However, concentric performance recovered within 10 min post exercise. The SSC performance curve showed a delayed depression 2 days after exercise. EMG activity during the SSC action showed a delayed depression, especially in the ankle extensors.

(3) Exhaustive SSC exercise induced a deterioration in the stiffness regulation mechanism characterized by a deficit in the the stretch reflex and subsequently in power in the SSC exercise. The delayed decrease in SSC performance was related to the possible delayed increase in muscle damage.

(4) The magnitude of the initial SSC fatigue influenced the subsequent time course of muscle mechanical performance. The delayed performance deficit in the SSC exercise was related to the depression in the initial stiffness and pre-activity of the working muscle accompanied by the altered pre-landing movement.

(5) During exhaustive SSC exercise, the neuromuscular control strategy was altered, so that the changed pre-landing joint kinematics influenced the subsequent post-landing joint kinematics and performance. The time-varying stiffness approach showed an interaction between pre-landing kinematics and post-landing stiffness regulation during fatigue. Altered post-landing stiffness regulation was related to delayed muscle damage.

6.1 Characteristics of SSC action

In the present study, a difference in performance recovery response was found between Max SSC and Max SJ after exhaustive SSC exercise (Fig. 10). The SSC action is characterized by active stretch (eccentric action) immediately followed by the concentric action (Komi 1992). Bosco et al. (1982b) have demonstrated that both reflex potentiation, which is presumably associated with improvement in stiffness and elastic energy, may operate effectively during SSC action. The significance of this reflex action on SSC performance has been discussed in detail (Komi and Gollhofer 1997). Therefore, the eccentric action plays an important role in regulating SSC performance. In addition, the lengthening contraction may induce selective recruitment of fast muscle and/or fast MU (Nardone and Schieppati 1988, Nardone et al. 1989). Moritani et al. (1990a) have demonstrated further the selective higher H-reflex gain in the "fast" gastrocnemius muscle as compared to "slow" soleus muscle during maximal hopping. It is likely therefore that the fast MU is selectively recruited in the SSC action. On the other hand, it has been reported that the motor unit recruitment pattern follows the size principle (Henneman et al. 1965) in the ballistic concentric contraction (Desmedt and Godaux 1977). Therefore, a different motor command and/or motor control strategy might contribute to the difference in fatigue response between the Max SSC and Max SJ after exhaustive SSC exercise. Considering the selective damage of the fast twitch fibres after the eccentric contraction (Jones et al. 1986, Fridén et al. 1983 1988a, Sjöström and Fridén 1984), the absence of a delayed decrease in the Max SJ performance seems to imply further the selective recruitment of relatively slow MU in the Max SJ as compared to the Max SSC. After SSC exercise, selective depressions in EMG activity were shown in the eccentric phase as well as pre-activation period before contact (Fig. 12). Therefore, the present results emphasize the functional significance of the pre-landing activity as well as eccentric action in stiffness regulation during the SSC action.

In this connection, knee extensor muscle-tendon stiffness during the eccentric phase in DJ could be explained by a combination of VL pre-activity and knee joint angular velocity at touchdown (Table 5). Pre-landing knee joint angular velocity could be used as a single measure of DJ performance (Fig. 8). In addition, DJ performance can be explained by a combination of the pre-landing knee joint angular velocity and knee extensor muscle-tendon stiffness. This combination is a better predictor than the combination of contact time and maximum rate of isometric force development (Max RFD). Therefore a possible connection between pre-landing activity and subsequent stiffness regulation and performance might be the factors regulating SSC performance.

However as already mentioned in the Methods section, the methodological limits of the present muscle-tendon model should be considered in interpreting these results. Individual muscle mass, training effect, type of sport activity and/or muscle fiber characteristics might also influence stiffness regulation.

In the landing activity such as occurred in the DJ, pre-activity has been considered to involve the centrally preprogrammed motor commands directing the required motor task (Dietz et al. 1981, Dyhre-Poulsen et al. 1991, Gollhofer and Kyröläinen 1991, Melvill Jones and Watt 1971a). Another function of pre-activation is assumed to be the increased sensitivity of the muscle spindle via enhanced α-γ co-activation to enhance the stretch reflexes, as suggested earlier (Gottlieb et al. 1981). The stretch reflex has a compensatory role in enhancing muscle stiffness after yielding during stretch (Allum

and Mauritz 1984, Hoffer and Andreassen 1981, Nichols and Houk 1976). Thus, improved stiffness regulation could be attained by proper pre-landing muscle activation. Because of the rather short duration before touchdown during the free fall period from a height of 50 cm which lasted 320 ms in the present DJ test, this pre-landing motion seems to be preprogrammed. Therefore the present results emphasize the additional functional significance of pre-landing activity in performing the SSC action more effectively in addition to the possible selective recruitment of fast MU.

6.2 Stiffness regulation during SSC exercise

6.2.1 Reflex-stiffness interaction

Allum and Mauritz (1984) have shown that the yielding of muscle stiffness during stretching is compensated by the short latency reflex. In addition, it has been pointed out that muscle stiffness represents the actual state in the contractile machinery of the muscle, that is filament overlap between the actin and myosin filaments (Ford et al. 1981), and is closely related to force production (Ford et al. 1986). Therefore, compensation in stiffness after stretching is thought to be associated with corresponding changes in muscle mechanical behavior, such as power production, which is a product of force and velocity. Along these lines, Kilani et al. (1989) have demonstrated decreased SSC performance by abolishing reflexes by drug injection. Thus, the stretch reflex during the eccentric action might be an important factor in regulating subsequent muscle stiffness, including concentric muscle mechanical performance during the SSC muscle action (Voigt et al. 1998).

Avela and Komi (1998) have estimated soleus peak muscle stiffness as the ratio of the estimated Achilles tendon force to the estimated soleus muscle length during the sledge jump. However, as suggested by Kirsch et al. (1993), the stretch reflex has time-varying and task-dependent characteristics which regulate posture and movement. Therefore instantaneous stiffness seems to be an appropriate measure for evaluating time-varying stiffness regulation during a short contact time SSC such as hopping and/or the DJ. Although Dyhre-Poulsen et al. (1991) have measured instantaneous stiffness change during the DJ, no attempt has been made to investigate the possible interaction between stiffness regulation and performance. In the present study, the decrease in the initial first peak of stiffness of the knee joint was related to the decrease in DJ performance after exhaustive SSC exercise ($r = 0.65$, $P < 0.05$) accompanied by a decrease in the short latency the stretch reflex M1 component of the VL muscle (Fig. 13). In addition, relative changes in stretch reflex M1 component of the VL muscle, knee positive peak power, contact time and the corresponding change in stiffness were closely coupled to each other (Fig. 14). These interrelationships emphasize the functional significance of the stiffness and short latency stretch reflex in regulating the subsequent power production and the whole movement during the SSC action. Thus the present results give additional confirmation of the possible interaction between the stretch reflex and stiffness regulation during a natural-type multijoint SSC action. It should be noted, however, that two subjects demonstrated the opposite trend in the upper two figures in Fig. 14. These individuals showed much higher VL M1, higher knee positive peak power and shorter contact time after exhaustive SSC exercise. Because DJ performance decreased in these cases, one could assume that the knee joint compensated for the function of other joints in these two

subjects as compared to the other subjects. Therefore the multijoint movement and/or neuromuscular control strategy was modulated after SSC fatigue in these individuals.

The first peak of stiffness and the stiffness changes during the VL M1 response decreased 2 h and 2 days after SSC exercise (Table 7) when a delayed increase in serum CK activity was observed. In the present study, these stiffness parameters seem to be related to the intrinsic property of muscle. Therefore it could be proposed that the delayed decrease in stiffness might be related to possible muscle damage associated with ultrastructural changes in muscle fibres (Fridén et al. 1988a).

6.2.2 Possible influence of muscle damage

Recent studies have revealed that exhaustive SSC exercises either of short duration with rebound jumps (Nicol et al. 1996) or longer lasting marathon running (Avela et al. 1999b) induce delayed muscle damage as well as reduced reflex sensitivity, which persists for several days after exercise. Because muscle spindle sensitivity was observed to change after the prolonged passive stretches (1 h) without any accumulation of blood metabolites such as lactate and serum CK, it could be assumed that exhaustive SSC exercise may induce not only extrafusal but also intrafusal fibre damage (Avela et al. 1999a). In addition, these alterations could influence stiffness regulation during the SSC action. In this connection, Avela and Komi (1998) have demonstrated that peak soleus stiffness decreases as measured indirectly after long-lasting marathon running. However, no information is available about the interaction between stiffness regulation and delayed muscle damage during the follow up period after exhaustive SSC exercise. In the present study, both the initial peak of stiffness and changes in stiffness during the VL M1 response decreased 2 days after exercise accompanied by a performance deficit and increased CK activity (Table 7). The present study therefore demonstrates the possible interaction between muscle damage and stiffness regulation associated with SSC performance during a follow up period after SSC exercise. As suggested by Nicol et al. (1996), these findings have been explained partly by reflex inhibition to the α-motoneuron pool, which could be mediated by the activation of the small diameter group III and IV afferents (Garland 1991). These small afferents are known to be activated by the chemical agents associated with muscle pain and inflammation after muscle damage (Mense 1977).

Recent studies have shown that the initial mechanical injury induces secondary injury by the delayed phagocytic response in which the force deficit is characterized by a bimodal trend during the follow up period after eccentric exercise (Faulkner et al. 1993, MacIntyre et al. 1996). A similar phenomenon has been observed after long-lasting SSC exercise (marathon run) in which the decreased stretch reflex M1 component as well as increased peak force reduction showed the bimodal pattern (Avela et al. 1999b). In the present study, a subject who showed a large performance deficit immediately after exercise (initial decline) showed early recovery 2 h after exercise followed by a subsequent decline in performance and delayed muscle damage 2 days after exercise (secondary decline, Fig. 15). Therefore the present results can be used as additional evidence that the magnitude of the initial mechanical injury is partly connected with the magnitude of the delayed muscle damage. However caution should be exercised in interpreting the serum CK results with respect to variability of individual response (Newham et al. 1983a) and lymphatic circulation in the removal of muscle protein (Stauber et al. 1990).

In the present study, the pre-activity of the VL muscle before contact DJ increased immediately after and 2 h after SSC exercise compared to the pre-fatigue value, which may be thought to compensate for the fatigue effect. However, the pre-activity of the VL muscle decreased 2 days and 4 days after compared to 2 h after, accompanied by the slow recovery of performance (Fig. 18). Therefore neural compensation might have been inhibited 2 days and 4 days after exercise. In this situation, the pre-landing motor command seemed to act to prevent further muscle damage at touchdown in the DJ. As the secondary injury progresses, the initial high impact load cannot be tolerated and the transfer of this impact force to the subsequent push-off phase becomes more difficult. In the present study, the delayed change (2H - 2D) in initial stiffness was negatively related to the delayed change in the knee joint angle at touchdown during DJ (Fig. 16). Therefore, the larger knee joint angle at touchdown, which might reflect the degree of the secondary injury, can be assumed to be related to the decrease in initial stiffness. As proposed in Fig. 17, it is possible that the damage induces modifications in the pre-landing motor control system, which could then influence the initial stiffness regulation after touchdown in the DJ. As suggested by Asmussen and Mazin (1978), activated signals from chemo- and mechano-receptors such as group III and IV afferents during fatigue could also be sent to the central nervous system (CNS), which then induces the development of the central inhibition. In other words, this phenomenon may suggest that the CNS sends inhibitory commands to prevent a powerful SSC action which would require a higher activated eccentric action. Interestingly, this model proposed to explain induced damage seems similar to the absorbing-type inefficient jump motion suggested by the non-fatigue measurement (Fig. 9). Therefore the motor control strategy may be modified to suit the inefficient absorbing-type movement by the delayed muscle damage. In this connection, lower spinal level excitability has been observed during the pre-landing phase in the absorbing-type DJ as compared to the bouncing-type DJ (Dyhre-Poulsen et al. 1991). This result might indirectly support the concept of damage-induced central inhibition of the movement strategy. Consequently, the damage-induced depression in SSC performance can be explained by the deterioration of the peripheral machinery as well as altered jumping strategy in relation to the central inhibition. Furthermore, the present results give rise the idea that exhaustive SSC exercise induces peripheral damage as well as possible central inhibition during movement.

6.2.3 Flexibility of the centrally preprogrammed motor control during SSC exercise

During exhaustive SSC exercise, joint kinematics, kinetics and EMG activity showed large alterations, especially in pre-landing joint kinematics (Fig. 21 and 22). Greater alterations in joint kinematics accompanied by acute changes in EMG activity during the pre-landing flight phase implies the greater sensitivity of the pre-landing activity to exhaustive SSC exercise as compared to post-landing activity. During the first half of the SSC exercise, eccentric force output (Fz *eb*) was kept at the same level by an increase in the EMG activity of the knee extensors. This force response is different from that found in animal experiments in which a greater force deficit occurred in the initial part of the whole contraction period under constant stimulation conditions (McCully and Faulkner 1986, Bentz et al. 1998, Lieber et al. 1991 1996). Therefore the constant eccentric force output associated with the concomitant increase in EMG activity clearly shows the existence of neuromuscular compensation in the natural human SSC.

During the second half of the exercise, alterations in joint kinematics were more evident in the contact phase of the SSC action (Fig. 21 and 22). A symmetrical relationship was shown between the increase in Knee ROM and decrease in Ankle ROM, which corresponds to the trade-off across the joint (Bonnard et al. 1994, Patla 1987). This alteration was coupled with decreased eccentric force output as well as increased contact time, which implies disturbance of the eccentric function. Therefore, the neuromuscular adaptation process is clearly switched to the push-off phase without any additional EMG support to the knee extensor muscles during the braking phase of the SSC. In the second half of the exercise, the pre-landing activity such as decreased VL pre-activity and increased ankle angular velocity during the downward period influenced the increasing post-landing Knee joint ROM (Fig. 23). The ankle flexion velocity while in pre-landing seems to correspond to increase in dorsiflexion followed by subsequent decrease in the Ankle ROM. Therefore the altered pre-landing motor control strategy affected the subsequent joint kinematics in the second half of the exercise. These adaptation patterns further demonstrate that the neuromuscular adaptation process varies within muscles, and across the joints and action phases of the SSC action.

In the present study, TVS and TVV were measured to evaluate time-varying stiffness. This procedure enables the detection of short range stiffness in the total leg system. Because short range stiffness can be influenced by prior muscle tension and movement (Joyce et al. 1974, Morgan 1977), it can be proposed that altered pre-landing activity might influence post-landing short range stiffness. In the second half of the exercise, increased pre-landing ankle flexion velocity was related to the reduction in short range stiffness (Stiff 1st in Fig. 5, 20 and 24). The reduced short range stiffness was further related to the subsequent reduction in performance as well as increased viscosity. Increased viscosity was related to delayed muscle damage. In general, short range stiffness has been considered to represent the initial attachment of the cross-bridges in the striated muscle (Flitney and Hirst 1978). Short range stiffness has also been identified in the intact human muscle-tendon complex regardless of tendon compliance (Hufshmidt and Schwaller 1987). Therefore the altered pre-landing joint kinematics might modulate the initial cross-bridge attachment which could further influence post-landing performance. Thus the present study indicates the chain-like serial interaction between the altered pre-landing motor control strategy, post-landing stiffness, post-landing performance and post-exercise muscle damage. In the present study, the short range stiffness can, however be referred to as ankle joint stiffness. Therefore, the present results emphasize the significant role the ankle joint function plays in regulating and/or coordinating the movement in the rebound-type of SSC action in terms of the transfer of mechanical energy (Prilutsky and Zatsiorsky 1994) as well as in its significant influence on total leg stiffness (Farley and Morgenroth 1999). The possible influence of foot placement at contact on subsequent SSC performance also confirms the significant function of the ankle joint (Kovács et al. 1999). The joint-specific motor control strategy might therefore be important for the multijoint SSC exercise.

In addition, progressive linear changes were observed in the knee joint angle at the end of stretch (Knee *eb*) accompanied by a corresponding linear change in pre-landing joint kinematics (Fig. 21). This alteration indicates that the knee bend, which corresponds to the absorbing-type inefficient jump motion (Fig. 9) and/or to muscle damage-induced inhibitory movement (Fig. 17), progressed immediately after the start of exercise. In this situation, the pre-landing motor control strategy also adapted

immediately to the alteration in the post-landing joint kinematics. These parallel alterations between pre- and post-landing joint kinematics further support the idea of the functional link between preprogrammed pre-landing motor control and post-landing performance. As suggested earlier by Asmussen and Mazin (1978), it could be proposed that the fatigue-induced peripheral disturbances might be signaled back to the CNS via group III and IV afferents which include chemo- and mechano-recepters. Therefore, CNS adapts flexibly to the functional requirement of the peripheral system.

Consequently, as proposed in Fig. 25, the present series of studies suggest the existence of fine tuning of the CNS to maintain optimum balance between the facilitation and inhibition of the peripheral system in terms of stiffness regulation during SSC exercise.

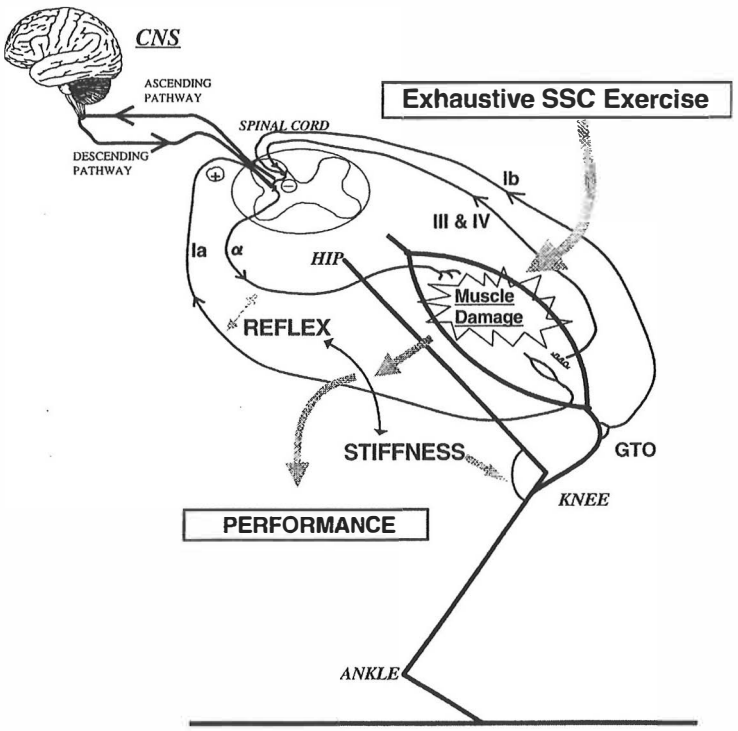

FIGURE 25 Proposed schematic diagram of the integrative motor control circuit in terms of stiffness regulation during SSC exercise.

7 PRIMARY FINDINGS AND CONCLUSIONS

The main findings and conclusions of the present study can be summarized as follows:

(1) Exhaustive SSC exercise induced a difference in fatigue response between the SSC action and the pure concentric action. The SSC action curve showed a long-lasting depression in pre-activity and in eccentric muscle function. These fatigue responses may be related to the selective recruitment of fast MU in the SSC action. SSC performance can be explained by the combination of pre-landing knee joint kinematics and knee joint muscle-tendon stiffness. The pre-landing activity associated with the selective recruitment of fast MU may characterize the SSC.

(2) After exhaustive SSC exercise, the decrease in initial knee joint stiffness was related to the corresponding decrease in SSC performance accompanied by a decrease in the stretch reflex M1 component of the VL muscle. There were close relationships between changes in knee joint stiffness, knee joint positive peak power, the stretch reflex of the VL muscle and contact time in the SSC action. Stretch reflex and stiffness were functionally coupled to regulate the multijoint SSC action.

(3) Initial joint stiffness showed a continuous depression during the follow up period after exhaustive SSC exercise accompanied by delayed muscle damage. Exhaustive SSC exercise induced long-lasting reduced stiffness regulation. Exhaustive SSC also induced mechanical injury which further influenced stiffness regulation. Initial mechanical injury, caused secondary injury which altered the SSC movement. A damage-induced inhibitory movement strategy took place after SSC fatigue.

(4) The neuromuscular control system alters its strategy immediately after the start of SSC exercise. As the exercise progresses neuromuscular adaptation occurs variously within the muscles, and across the joints and action phases of the SSC action. Therefore the neuromuscular control strategy is operated flexibly and comprehensively during the course of SSC exercise to meet the functional requirements of the peripheral system.

Pre-landing ankle joint kinematics influence the post-landing joint kinematics as well as initial leg stiffness. Altered initial leg stiffness affect the subsequent performance and delayed muscle damage. Ankle joint plays an important role in the stiffness regulation during SSC exercise.

As a consequence, CNS maintains optimum balance between fascilitation and inhibition of the peripheral system in terms of stiffness regulation during SSC exercise.

8 YHTEENVETO

Ihmisen luonnollisessa liikkumismallissa lihaksen voimia tuotetaan venymis-lyhenemissyklin (SSC) tavoin. SSC:ssa aktiivinen lihas ensin venyy ja tätä venytysvaihetta seuraa välittömästi lyhenemistoiminta. Lyhenemistoiminta eli konsentrinen lihassupistus on SSC:n yhteydessä erittäin tehokas ja taloudellinen verrattuna tilanteeseen, jossa lyhenemistoiminta aloitetaan ilman aktiivista esivenytysvaihetta.

SSC hyödyntää normaalin aineenvaihduntatoiminnan lisäksi erityisesti lihaksen jäykkyyssäätelyyn liittyviä komponentteja. Lihaksen jäykkyyssäätelyssä on refleksitoimintojen merkitys huomattava, mutta niiden kvantifiointi on ollut tutkimusmenetelmien rajallisuuden vuoksi erittäin vaikea. Huolimatta eläinkokeilla tuotetun runsaan tiedon määrästä, jäykkyyssäätelyn todellinen mekanismiketju on ollut vaikea ymmärtää luonnollisen lihastoiminnan, SSC:n kannalta. Tilanne on erityisen ongelmallinen sellaisen SSC:n yhteydessä, jossa liike tapahtuu monen nivelen kautta.

Uuvuttavaa, SSC-tyyppistä lihasväsymystä on viime aikoina tutkittu suhteellisen paljon ja tulokset näyttävät voittopuolisesti osoittavan, että lihaksen refleksitoiminta häiriintyy tällaisessa tilanteessa. Lihaksen proprioseptiset refleksit, erityisesti lihassukkulasta ja jänteistä lähtevät refleksiohjaukset, ovat osa lihaksen jäykkyyssäätelyä. Tällöin on syytä olettaa, että SSC-väsymismalli käytettynä normaalia, väsymistä aiheuttamattoman SSC:n yhteydessä, auttaisi ymmärtämään koko jäykkyyssäätelyn mekanismia.

Tämän tutkimussarjan tarkoituksena oli selvittää millä tavoin jäykkyyssäätelyn eri tekijät adaptoituvat siirryttäessä normaalitilanteesta uuvuttavaan SSC-väsymiseen ja siitä palautumiseen. Tutkimussarja koostuu viidestä osakokonaisuudesta. Näissä kaikissa koehenkilöinä oli miehiä, jotka suorittivat SSC-tyyppisiä koko alaraajan ojentajalihasketjun toimintoja. Tutkimuslaitteet olivat moderneja hermo-lihastoiminnan mittaukseen käytettäviä sekä elektromyografian (EMG) että lihasmekaniikan mittalaitteita. Ensimmäisessä tutkimussarjassa selvitettiin niitä hermo-lihastoiminnan tekijöitä, jotka ovat yhteydessä hyppelytyyppisen SSC-toiminnan säätelyyn erityisesti lattiakontaktia edeltäen (esiaktiivisuusvaihe) ja kontaktin aikaisen vaiheen yhteydessä. Toinen tutkimusvaihe selvitti tarkemmin SSC-toiminnan sisäistä väsymismallia ekshaustiivisen SSC-kuormituksen yhteydessä. Erityisenä tavoitteena oli selvittää, miten eksentrinen lihastoimintavaihe (esivenytys) kuormittuu ja väsyy suhteessa konsentriseen vaiheeseen. Tutkimussarjan kolmas vaihe selvitti SSC-väsymisen yhteydessä esiintyneiden palautuvien lihasvaurioiden vaikutusta lihastoiminnan

jäykkyyssäätelyyn. Uuvuttava SSC-kuormitus on erinomainen keino aiheuttaa lihasvaurioita, jotka esiintyvät kahdentyyppisinä ilmiöinä: välitön vaurioitumisvaihe ja 1-2 päivän kuluttua esiin tuleva sekundaarinen vauriovaihe. Neljännen tutkimusvaiheen tarkoituksena oli selvittää millä tavalla lihasvauriomekanismit ja niiden vaiheet ovat yhteydessä lihasmekaniikassa ja lihaksen jäykkyydessä tapahtuviin muutoksiin. Viides ja viimeinen tutkimusvaihe keskittyi SSC-väsytyksen ja siihen liittyvän lihasvaurion aiheuttamia lihastoiminnan kontrollia säätelevien tekijöiden selvittämiseen. Erityisenä tavoitteena oli paikantaa niitä tekijöitä, jotka liittyvät esiohjelmoidun motorisen kontrollin muutoksiin suhteessa nivelen kinetiikan ja jäykkyyssäätelyn muutoksiin uuvuttavan SSC-kuormituksen aikana.

Tutkimussarjan tulokset on julkaistu kuutena erillisenä julkaisuna, jotka ilmestyvät alan kansainvälisissä tieteellisissä lehdissä. Seuraavat kuusi päälöydöstä ovat huomattavia tämän tutkimussarjan yleistettävyyden kannalta:

(1) Uuvuttava SSC kuormitus aiheutti voimakkaamman väsymisvaikutuksen spesifisesti eksentrisessä vaiheessa. Tämä näkyi esiaktiivisuuden sekä eksentrisen tehon pitkäaikaisena heikkenemisenä. Löydös on todennäköisesti yhteydessä valikoivaan nopeiden lihassolujen rekrytoitumiseen SSC:n yhteydessä.

(2) Lihaksen jäykkyyssäätelyn osalta erityisesti venytysrefleksin amplitudi reisilihaksessa oli alentunut ekshaustiivisen SSC-kuormituksen jälkeen. Tämä refleksitoiminnan heikkeneminen oli puolestaan kiinteässä yhteydessä polvinivelen jäykkyyden ja tehon kanssa.

(3) Niveljäykkyyden depressio venymisvaihetta seuraavan seurantajakson (yksi viikko) aikana oli riippuvainen tuotetun lihasvaurion asteesta. Lihasvaurio aiheuttaa inhibitorisen liikestrategian.

(4) Hermolihastoiminnan strategia muuttuu SSC-väsytyksen aikana vaihdellen lihasten välillä, nivelten välillä sekä SSC-toiminnan eri vaiheiden suhteen. Tämä havainto ilmentää kontrollitoiminnan spastisuutta ja valmiutta sopeutua ulkoisen kuormituksen aiheuttamiin vaikutuksiin.

(5) Hyppelytoiminnassa esikontaktivaiheen jäykkyyden aste vaikuttaa lihas- ja niveljäykkyyteen kontaktivaiheen aikana. Samalla sillä on merkitystä lihasvaurion voimakkuuteen toistuvien SSC-suoritusten yhteydessä.

(6) Keskushermoston osuus on huomattava SSC-kuormituksen yhteydessä. Se ylläpitää periferisessä järjestelmässä esiintyviä inhibitorisia ja fasilitatorisia toimintoja tasapainossa, jotta lihaksen jäykkyyssäätely vastaa ulkoisen kuormituksen edellyttämiä vaikutuksia.

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10 APPENDIX

In this model

$$T = K_2 \cdot \Delta\theta_2 = K_1 \cdot \Delta\theta_1 + B_1 \cdot \dot{\theta}_1 + T_1 \quad \text{---------- (1)}$$

$$\Delta\theta = \Delta\theta_1 + \Delta\theta_2 \quad \text{---------- (2)}$$

$$T = K_1(\Delta\theta - \Delta\theta_2) + B_1(\dot{\theta} - \dot{\theta}_2) + T_1$$

$$= K_1\left(\Delta\theta - \frac{T}{K_2}\right) + B_1\dot{\theta} - B_1\frac{\dot{T}}{K_2} + T_1$$

according to McMahon, (1984), equation (1) is extended as follows using equation (2)

$$\left(1 + \frac{K_1}{K_2}\right)T + \frac{B_1}{K_2}\dot{T} = K_1\Delta\theta + B_1\dot{\theta} + T_1$$

$$T + \left(\frac{B_1}{K_1 + K_2}\right)\dot{T} = \left(\frac{K_1K_2}{K_1 + K_2}\right)\Delta\theta + \left(\frac{K_2B_1}{K_1 + K_2}\right)\dot{\theta} + \left(\frac{K_2T_1}{K_1 + K_2}\right) \quad \text{----------(3)}$$

then

replace

$$\frac{B_1}{K_1 + K_2} = A \quad \frac{K_1K_2}{K_1 + K_2} = C \quad \frac{K_2B_1}{K_1 + K_2} = D \quad \frac{K_2T_1}{K_1 + K_2} = E$$

then equation (3) is expressed as a simple form as follows

$$T = -A\dot{T} + D\dot{\theta} + C\Delta\theta + E \quad \text{----------(4)}$$

in equation (4), variables $T, \dot{T}, \dot{\theta}, \Delta\theta$ are known

parameters *A, D, C, E* can be obtained by the multiple regression analysis

final solutions are as follows

$$K_1 = \frac{CD}{D - AC} \quad K_2 = \frac{D}{A} \quad B_1 = \frac{D^2}{D - AC} \quad T_1 = \frac{ED}{D - AC}$$

ORIGINAL PAPERS

I

Stretch shortening cycle fatigue: interactions among joint stiffness, reflex, and muscle mechanical performance in the drop jump

by

Tomoki Horita, Paavo V. Komi, Caroline Nicol and Heikki Kyröläinen

Eur J Appl Physiol 73: 393-403, 1996

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ORIGINAL PAPERS

II

Effect of exhaustive stretch-shortening cycle exercise on the time course of mechanical behaviour in the drop jump: possible role of muscle damage

by

Tomoki Horita, Paavo V. Komi, Caroline Nicol and Heikki Kyröläinen

Eur J Appl Physiol 79: 160-167, 1999

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III

Interaction between prelanding activities and stiffness regulation of the knee joint musculoskeletal system in the drop jump: implications to performance

by

Tomoki Horita, Paavo V. Komi, Caroline Nicol and Heikki Kyröläinen

(submitted)

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IV

Exhausting stretch-shortening cycle (SSC) exercise causes greater impairment in SSC performance than in concentric performance

by

Tomoki Horita, Paavo V. Komi, Ismo Hämäläinen and Janne Avela

(submitted)

https://doi.org/10.1007/s00421-002-0716-z

V

Changes in neuromuscular performance during repeated stretch-shortening cycle (ssc) exercise I: adaptation patterns in the joint kinematics and electromyographic activities

by

Tomoki Horita, Paavo V. Komi, Ismo Hämäläinen and Janne Avela

(submitted)

CHANGES IN NEUROMUSCULAR PERFORMANCE DURING REPEATED STRETCH-SHORTENING CYCLE (SSC) EXERCISE I: ADAPTATION PATTERNS IN THE JOINT KINEMATICS AND ELECTROMYOGRAPHIC ACTIVITIES

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Abstract

Exhaustive repeated stretch-shortening cycle (SSC) exercise was carried out to investigate the neuromuscular adaptation process during SSC exercise with a special focus on the pre-landing period of SSC action. The results demonstrated clear alterations in the adaptation in the neuromuscular function revealed by joint kinematics and surface electromyographic (EMG) activities. Changes in kinetics and kinematics during exhaustive SSC fatigue were characterized by a mean 27 % increase of the contact time. This was associated with a 14 % increase of the range of motion of the knee joint during braking phase (Knee ROM) and a 18 % decrease in eccentric force output as judged by the force level at the end of braking phase (Fz *eb*). Relative analysis of the contact time, Knee ROM and Fz *eb* revealed no change during the first half of the SSC exercise. This was accompanied by progressive linear increase in knee extensor EMGs during the braking phase (VL +26 %, VM +17 %) suggesting that the adaptation process would occur mainly in the braking phase. The second half of the exercise was characterized by increase of the contact time and Knee ROM as well as decrease in Fz *eb*. EMG activities showed additional increase in knee extensor EMGs in the push-off phase (VL +18%, VM +6 %) with no additional increase in the braking phase. The adaptation process seemed therefore to occur mainly in the push-off phase during the second half of the exercise. In this part of the exercise, the changes in Knee ROM were related with changes in the VL pre-activity (r = -0.74, $P < 0.05$) and pre-landing ankle flexion velocity (r = 0.82, $P < 0.05$). These interactions suggest the possible influence of central adaptation as revealed by altered pre-landing activities on the subsequent SSC performance during second half of the exercise.

1. Introduction

It has been suggested that exhausting stretch-shortening cycle (SSC) exercise induces selective depression of SSC performance associated with decreased stiffness regulation and delayed muscle damage [21, 22] as well as reduced reflex sensitivity [5, 31]. Two major hypotheses such as presynaptic reflex inhibition from group III and IV afferents [17] and disfacilitation of α-motoneuron pool [7] have been presented to explain the delayed mechanism of the reduced reflex sensitivity. It has been known that group III and IV afferents could be activated by a metabolic fatigue [36] and by some chemical agents associated with muscle damage [30]. Recent studies have therefore emphasized the role of presynaptic inhibition via group III and IV afferents as well importance of the reduction in the activity of the large-diameter group Ia afferents in the muscle spindle in situations, where severe delayed muscle damage occurs after strenuous SSC exercise
[4, 5].

In addition to these neural modifications, exhausting SSC exercise has been shown to induce modification in the whole jumping kinematics and kinetics in the contact period of SSC exercise. A reduced tolerance to impact, as indicated by a fast drop in the vertical ground reaction force after the initial force peak, has been reported both after arm [18] and leg [32, 5] SSC exercise. Furthermore, joint kinematics in the SSC exercise has been shown to be modified for several days during which a delayed increase in muscle inflammation and in muscle pain take place [21, 22]. These modifications could also be interpreted as resulting from changes in the "strategy" to perform the SSC, i.e. in the neuromuscular adaptation to the impaired subsequent SSC

action.

In this line, possible interaction between pre-landing motor control and subsequent performance of SSC after exhaustive SSC type exercise has been discussed [22]. It could also be supposed that neuromuscular strategy might be modified during the time course of fatiguing SSC exercise to perform a required task [33]. Bonnard et al. [8] have demonstrated the trade-off mechanism between muscles within a joint and between muscles across joints to compensate the fatigue effect of repeated single joint SSC during contact period of e.g. submaximal hopping. Altered lower limb segment kinematics [6, 13, 14] and increased contact time during the stance phase [32] have been characterized as fatigue effects during running. However, no previous study has examined the precise neuromuscular adaptation process involving pre-landing period during multijoint SSC exercise such as drop (rebound) jump in which the larger muscle groups of the lower limb are activated. Therefore the present study was undertaken to investigate the neuromuscular adaptation process during exhaustive SSC exercise of the lower limb segments with a special reference to pre-landing period of SSC. For this purpose, repeated submaximal rebound jumps on a sledge ergometer was used to induce exhaustive SSC fatigue. This exercise systematically induces muscle damage and pain and could lead to intermuscular and interarticular compensatory adaptation in the course of the SSC exercise and during its recovery [21, 22, 31].

2. Methods

2.1. Protocol

Eight healthy male volunteers gave their written consent to participate in this study. Their mean age, stature and body mass were 28 ± 4 years, 180 ± 4 cm and 76 ± 5 kg, respectively. The subjects were well informed about the possible risks associated with the experiment. The study was approved by the University Ethical Committee.

2.2. Experimental design and fatigue protocol

The fatigue protocol and analytical methods have been fully described in previous reports [21, 22, 31]. Fatigue was induced by an exhausting SSC exercise on a sledge apparatus which was inclined at 23 degrees from horizontal and equipped with a force plate (natural frequency: 200 Hz). The details of this apparatus have been described previously [3, 24, 26]. The subjects performed a series of exhaustive bilateral submaximal rebound jumps along the gliding track of the sledge. The subjects were instructed to rebound as long as possible to a height which was set at 70 % of their pre-determined maximal height (Fig. 1). In the present study, the average dropping height was 64 ± 6 cm. The exercise session was stopped when the subject could not reach the submaximal rebound height. On the average, the subjects performed 87 ± 24 repetitions, which corresponded a 2.5 ± 0.7 minutes durations. To examine the fatigue effects on SSC muscle function, a jump test was performed on the sledge apparatus before and after the SSC fatigue exercise as well as 2 days and 4 days after. Blood samples were also taken in each test session to measure the lactate (LA) and serum creatine kinase (CK) activity.

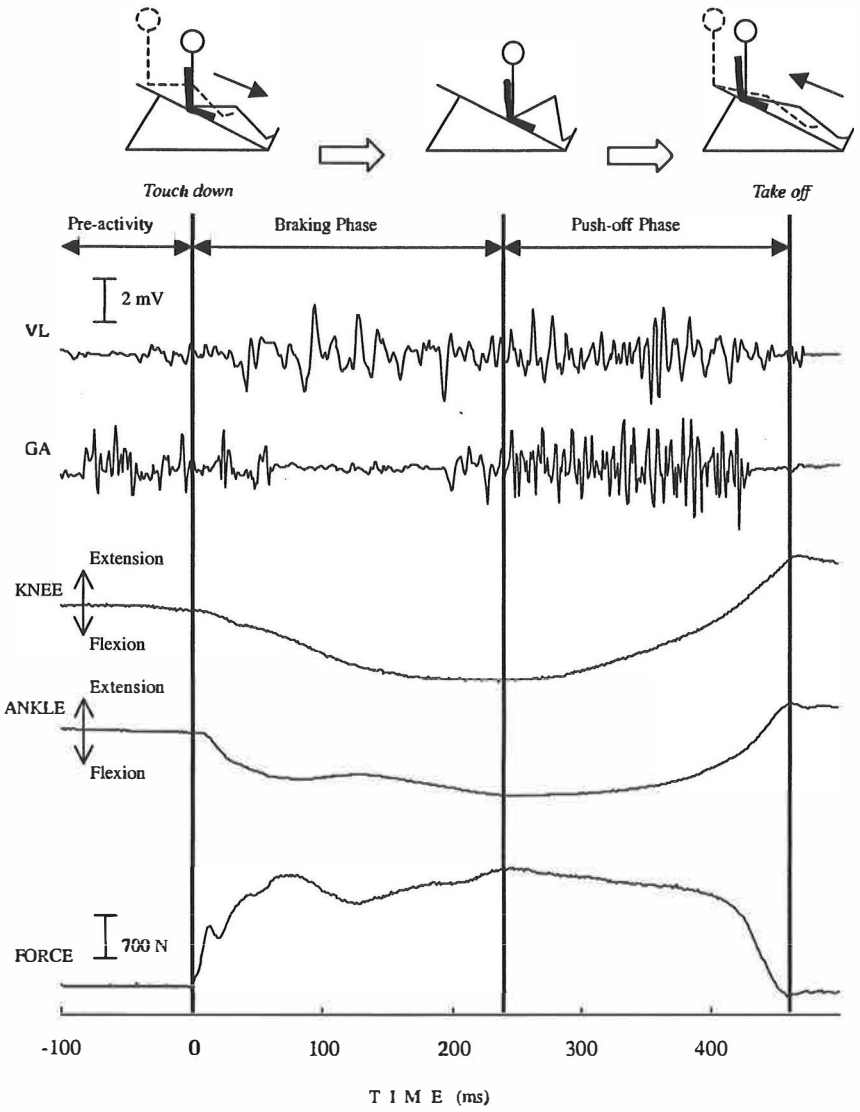

FIGURE 1 Typical record of the stretch-shortening cycle (SSC) sledge jump. Upper illustration shows the subject's movement on the sledge. Lower records, from top to bottom, indicate the vastus lateralis (VL) electromyographic (EMG) activity, gastrocnemius (GA) EMG activity, knee joint angle, ankle joint angle and reaction force.

2.3. Jump test

In each testing session, the jumping measurement consisted of a vertical drop jump from a pre-determined height (Max SSC), in which lower limb extensors are actively stretched after touch down before the subsequent concentric action. Max SSC measurements were performed before and 20 min, 2 days and 4 days after the SSC fatiguing exercise. In the present study, the maximal rebound height of jump was considered as an index of the jumping performance.

2.4. Electromyographic activity during jumping

Surface electromyographic (EMG) activities from the vastus lateralis (VL), vastus medialis (VM), medial gastrocnemius (GA) and soleus (SO) muscles of the right leg were recorded using miniature surface bipolar electrodes (Beckman skin electrode 650437, USA) and transmitted telemetrically (Glonner, Germany) during fatiguing exercise and jump test. The electrodes were placed longitudinally over the muscle belly with an interelectrode distance of 20 mm. Care was taken so that the interelectrode resistance was below 2 kΩ. The electrode positions were carefully marked on the skin to ensure the same electrode position in each test throughout the whole experiment. The EMG signals were stored simultaneously with the force plate signal and the goniometric signals recorded from the knee and ankle joint on a computer via a real time data acquisition system (Codas, Dataq Instruments Inc., USA), which included a 12-bit A/D converter with a sampling frequency of 1 kHz.

A cross-correlation analysis [37] was used to quantify possible cross-talk between adjacent muscles during the DJ performed by four subjects. The average cross-correlation coefficient (r_{xy}) between VL and VM muscles was 0.10 ± 0.06 ($P > 0.05$). Furthermore, GA and SO muscles also showed a low cross-correlation coefficient (r_{xy} = 0.18 ± 0.12, $P > 0.05$). Therefore, it was assumed that the extent of cross-talk was negligible in the present study.

EMG was integrated and then time normalized to calculate average EMG activity (aEMG) in the following three time periods; pre-activation before contact, braking phase (eccentric) and subsequent push-off phase (concentric). The pre-activation phase was defined as 100 ms preceding ground contact [25](Fig. 1).

2.5. Kinetic and Kinematic analysis during fatiguing rebound jumps

In the ground reaction force curve of rebound jump, first peak value (Fz 1^{st}) and force level at the end of the braking (eccentric) phase (Fz *eb*) were analyzed. It has been shown that the larger force output can be achieved at the starting point of concentric phase (end of eccentric phase) in SSC as compared to pure concentric and isometric contraction [2]. Therefore Fz *eb* was considered to evaluate the efficacy of SSC action. In the knee joint kinematics, peak flexion angle during the flight period, range of motion during the braking phase (Knee ROM) and the knee bending (flexion) at the end of braking phase (Knee *eb*) were analyzed. In the ankle joint, pre-landing angular velocity during the downward period of the flight phase and range of motion during braking phase (Ankle ROM) were analyzed. The average curves of the ground reaction force and joint angles during the course of the SSC exercise are shown in Fig. 2. In the present study, the time course of the SSC exercise was divided into ten relative stages (initial stage, 10% – last stage, 100%) based on the individual total number of jumps achieved during exercise. From each stage, ten successive jump records were analyzed for each subject (n=8). The group mean at each stage of the SSC exercise was then calculated for a total of 80 samples.

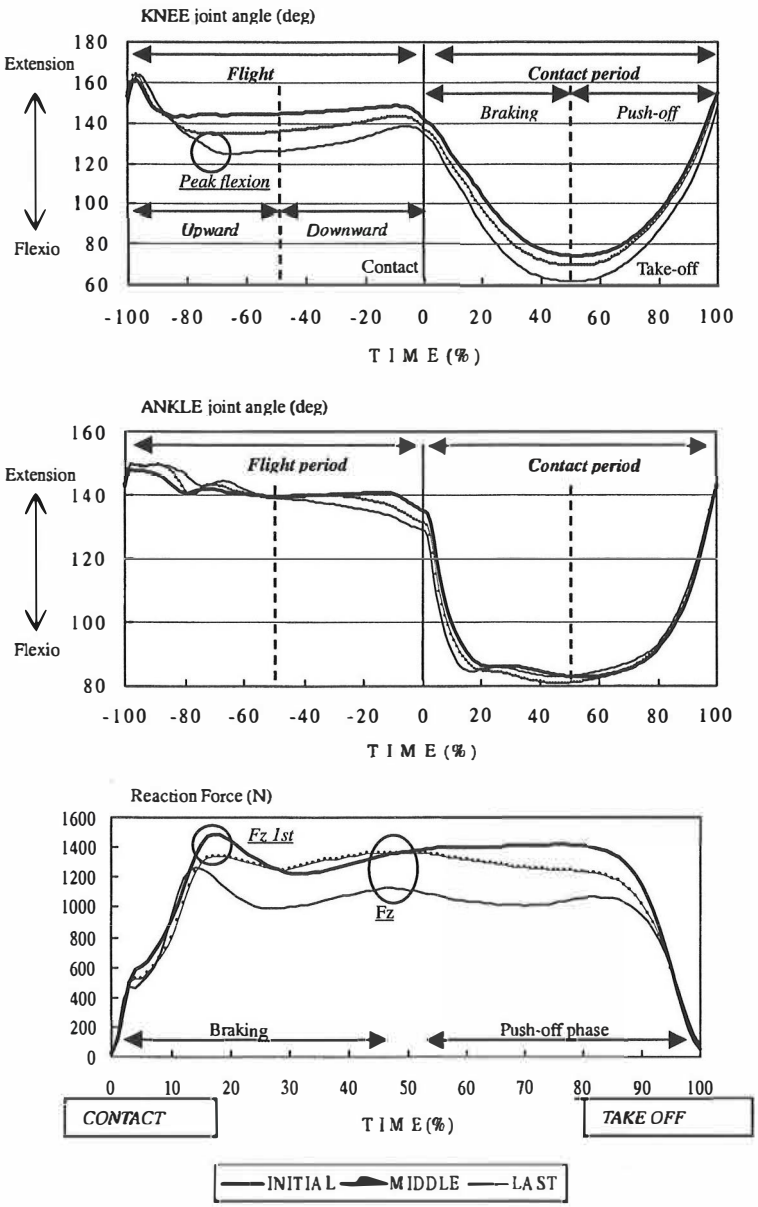

FIGURE 2 Average curves of the knee joint angle (*top*), ankle joint angle (*middle*) and reaction force (*bottom*) in each stage of the SSC exercise (*bold line*, initial stage; *thin line with circle dot*, middle stage; *thin line*, last stage). Explanation of each stage see text. Contact period normalized as 100 %. Flight period normalized as –100 %. In the knee joint angle, peak flexion angle is denoted as circle. In the reaction force, first peak and force level at the end of braking phase are denoted as circle.

2.6. Blood analysis

Blood lactate concentration was determined at rest, immediately after the sledge exercise as well as 3, 5 and 30 min later using a lactate analyzer (model 640, Roche). The serum CK activity was measured at rest and 30 min after the sledge exercise as well as 2 days and 4 days after exercise. The CK activity was analyzed using a CK ultra-violet test kit (Boehringer Mannheim, Germany).

2.7. Statistics

Time varying changes of the parameters during exercise were tested by an analysis of variance (ANOVA) for repeated measurements on one factor. Then a post-hoc LSD multiple comparison was performed. Pearson's correlation coefficients were calculated to examine the relationship between the relative changes of the different parameters. Statistical calculations were performed with a statistical package (STATISTICA ver. 4.5, StatSoft, USA). The level of significance was set at $P < 0.05$.

3. Results

3.1. Max SSC performance, Blood LA and CK changes

Blood LA concentrations increased significantly 3 min (6.8 ± 1.1 mmol·l^{-1}, P<0.001) and 5 min after (7.1 ± 0.8 mmol·l^{-1}, P<0.001) the SSC exercise bout, as compared to the pre-exercise value (1.5 ± 0.3 mmol·l^{-1}). Serum CK activity was markedly elevated on the 2nd day (477 ± 272 U·l^{-1}, P<0.001) and on the 4th day (373 ± 172 U·l^{-1}, P<0.05) as compared to the pre-exercise levels (211 ± 41 U·l^{-1}). As compared to pre-fatigue performance (91.8 ± 8.0 cm), the Max SSC rebound height was significantly reduced on the 2nd day (85.8 ± 4.9 cm, P<0.01) but not in 20 min (89.3 ± 7.7 cm) and 4th day (88.8 ± 9.4 cm). Therefore, the present SSC exercise induced acute metabolic fatigue and delayed possible muscle damage accompanied with delayed decrease in SSC performance.

3.2. Changes in mechanical performance during repeated SSC exercise

Contact time in each rebound jump increased progressively from the initial stage (554 ± 90 ms) to the last stage (702 ± 100 ms, P<0.001) of the exercise. Downward displacement of the sledge during the braking phase of the rebound jump also increased from 39 ± 6 cm (initial stage) to 45 ± 7 cm (last stage, P<0.001). Conversely, Fz 1st decreased from 1581 ± 213 N (initial stage) to 1310 ± 269 N (last stage, P<0.001) as well as Fz *eb* (from 1348 ± 278 N to 1085 ± 33 N, P<0.001). As shown in Fig. 2, the averaged force-time curve clearly showed the depression of force output in the last stage of the exercise as compared to the initial stage. The decreased force output was therefore compensated by increased contact time to achieve the same external work output.

3.3. Changes in joint kinematics during repeated SSC exercise

Average curves of the ankle and knee joint angles are shown in Fig. 2. During the flight

period, the maximal knee joint flexion decreased progressively from 141 ± 13 deg to 121 ± 16 deg (P<0.001) meaning that the knee was more extended towards the final stage of exercise. As shown by the increased ankle flexion velocity before contact (0.39 ± 0.34 rad·s^{-1}, P<0.001) as compared to the initial stage (0.17 ± 0.23 rad·s^{-1}), this joint was placed in a more flexed position during the downward period of the flight phase at the last stage of the SSC exercise. During the braking phase of the contact period, Knee ROM increased from 68 ± 12 deg to 74 ± 9 deg (P<0.001). This was accompanied by a decrease in the Knee *eb* (from 75 ± 15 deg to 61 ± 11 deg, P<0.001). Conversely, Ankle ROM decreased during the contact phase from 52 ± 5 deg to 46 ± 8 deg (P<0.001). Thus the joint kinematics of the ankle and knee joints were modified during exhaustive SSC exercise. These modifications were seen in both flight period and contact period of the SSC exercise.

3.4. Pattern of the changes in kinetics and kinematics during the time course of the SSC exercise

Relative changes in kinetics and kinematics during the time course of the SSC exercise are shown in Fig. 3. During the flight period, both ankle joint flexion ROM and knee joint extension ROM increased linearly immediately during the early stage of SSC exercise. After the initial stage (30 % of the total number of the jumps), ankle joint flexion ROM followed a plateau before presenting a secondary increase prior to the last stage of the exercise (80 % of the total number of the jumps) until exhaustion. However, the knee joint extension ROM showed uniform increase until prior to exhaustion except for the middle stage of the SSC exercise (50 % of the total number of the jumps).

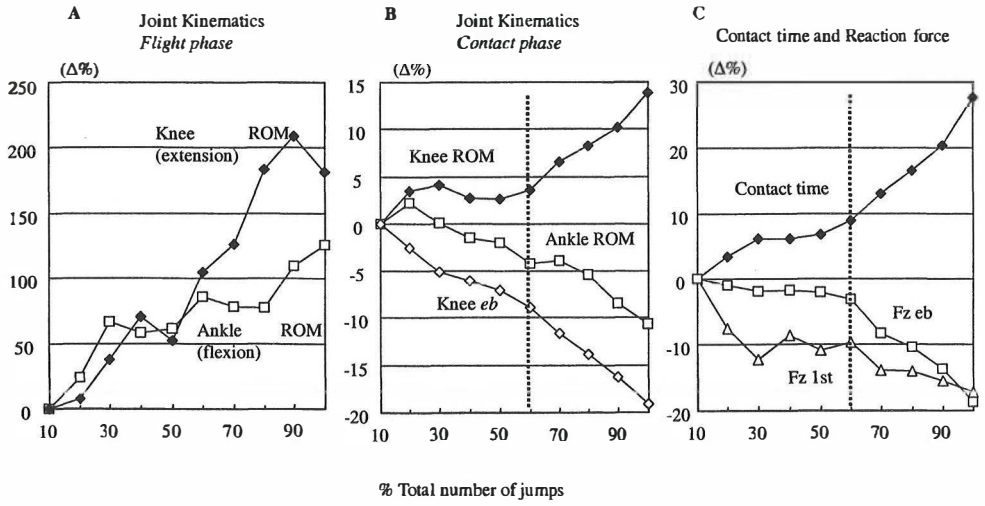

FIGURE 3 Relative changes in kinetics and kinematics during time course of the SSC exercise. A: joint kinematics during flight phase. B: joint kinematics during contact phase. C: contact time and reaction force. ROM; range of motion. Knee *eb*: knee joint angle at the end of braking phase. Fz *eb*: force level at the end of braking phase. Fz 1st: first peak of reaction force. Horizontal axis indicates the total number of jumps as relative value.

During contact period, Knee *eb* showed linear decrease until exhaustion. On the other hand, Knee ROM showed a bimodal pattern that consisted of initial small increase and subsequent plateau until the middle stage of the exercise (60 % of the total number of jumps) followed by a secondary large increase until the last stage of the exercise. Ankle ROM also showed a similar pattern with the knee joint but in opposite direction. Therefore, the Knee ROM and Ankle ROM changed proportionally in opposite directions during contact phase of the rebound jump throughout the time course of the fatiguing exercise.

Changes in contact time and Fz *eb* demonstrated similar patterns as observed in Knee ROM and Ankle ROM during the contact phase. Fz 1^{st} showed initial depression to the 30 % of the total number of jumps. Thus, repeated exhaustive SSC exercise induced kinematic and kinetic changes during the SSC exercise, especially in the kinematics of the flight phase during which the changes were approximately 10 - 20 times greater than those observed during the contact period.

3.5. Changes in aEMG activities

Pre-activity of the measured four muscle groups (SO, GA, VL and VM) showed the same significant trend of decreasing activity during the last stage of the SSC exercise as compared to the initial stage (SO: -14%, GA: -31%, VL: -22%, VM: -34%, $P<0.001$). Both GA and VM muscle pre-activities showed greater reduction as compared to SO and VL muscles. During the braking phase of the contact period, knee extensors (VL and VM) showed increased aEMG activity during the last stage of exercise (VL: +26%, VM: +18%, $P<0.001$). Whereas the triceps surae aEMG activity decreased (SO: -16%, GA: -24%, $P<0.001$). This was also the case during the push-off phase, in which the aEMG activity of knee extensor muscles increased (VL: +18%, $P<0.001$, VM: +6%, NS) despite a concomitant decrease in triceps surae aEMG activity (SO: -15%, GA: -34%, $P<0.001$). Thus, the triceps surae aEMG activity decreased continuously, from the pre-activation phase to the push-off phase through the braking phase, during the course of the SSC exercise. The largest reduction was observed in the GA muscle.

3.6. Pattern of the changes in the aEMG activities

Relative changes in the aEMG activities are shown in Fig. 4. In the pre-activation phase, all the muscles (SO, GA, VM and VL) showed an initial depression during the first 20 % of the jump series. Thereafter, these muscles showed individual responses. GA activities showed continuous decrease until exhaustion. SO activities showed further small decrease until middle stage of the SSC exercise (50 % of the total number of jumps) followed by a compensatory increase until the last stage of exercise. VL activities showed same activation level until exhaustion despite the large deviations. VM activities showed continuous decrease until exhaustion that was rather similar to those of GA activities.

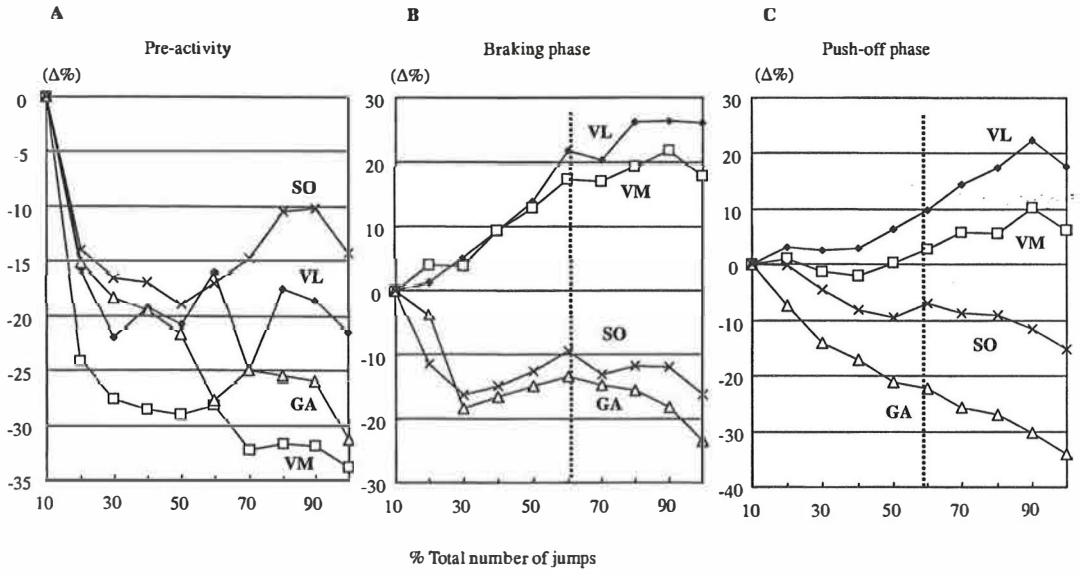

FIGURE 4 Relative changes in EMG activities of the VL, vastaus medialis (VM), GA and soleus (SO) muscles during time course of the SSC exercise. A: pre-activity. B: braking phase. C: push-off phase. Horizontal axis has same scale as in Fig. 3.

In the braking and push off phases, the knee extensor muscles (VL and VM) increased their EMG activities throughout the exercise. The opposite decrease of EMG activity was seen in the triceps surae muscles (SO and GA). Fig. 4 shows in detail the patterns of these changes.

3.7. Relationship between pre-landing kinematics and post-landing kinematics

As shown in Fig. 3 and 4, a turning point of the fatigue response occurred after the middle stage of the exercise (60 % of the total number of jumps) as observed in the joint kinematics (Knee ROM and Ankle ROM), contact time and kinetics (Fz *eb*) during contact period of the rebound jump. Therefore, another adaptation process might have dominated during the last 30 % of the jump series. At this stage, decrease in pre-activity of VL muscle and increase in ankle flexion velocity during downward period were related with the increase in Knee ROM (Fig. 5 A, B). Increase in the Knee ROM was also related with the increase in the contact time (Fig. 5, C). Therefore, the pre- and post- landing kinematics and over all performance were interrelated at the 70 % of the total number of jumps.

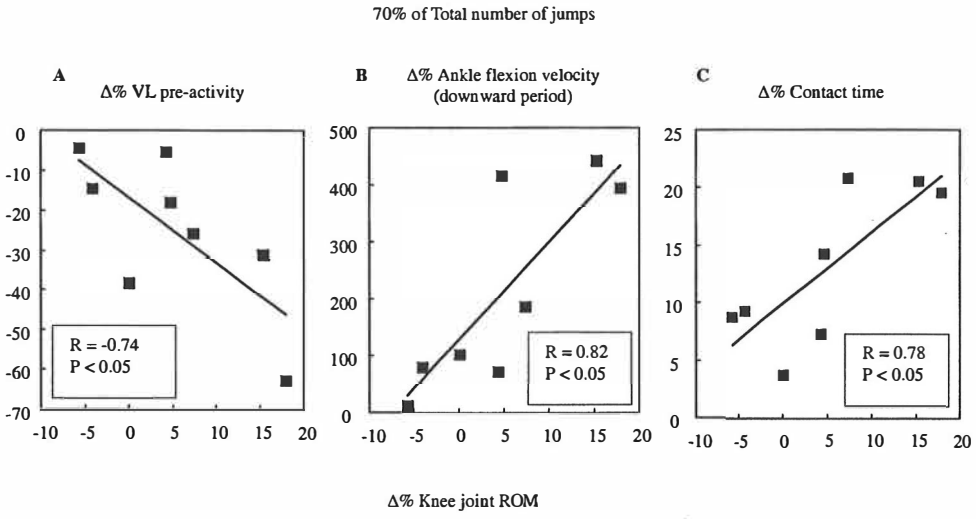

FIGURE 5 Correlation between changes (Δ) in range of motion of the knee joint and VL pre-activity (A), ankle flexion velocity during downward period (B) and contact time (C). All values were collected at second half of the SSC exercise (70 % of total number of jumps).

3.8. Relationship between muscle length change during exercise and post-exercise performance

As shown in Fig. 3, Knee *eb* demonstrated linear decrease until exhaustion that implies progressive increase in the knee bend during the course of the exercise. Increase in the knee bend at the last stage of the exercise (90 % of the total number of the jumps) was related with the delayed decrease in the post-exercise SSC performance on 2 days after the exhaustive SSC exercise (Fig. 6, A). Increase in the knee bend was further related with the delayed increase in the CK activities on 4 days after the exercise (Fig. 6, B).

4. Discussion

As expected, exhaustive SSC exercise induced potent alterations of joint kinematics, kinetics as well as EMG activities. This alteration was especially clear in the kinematics of the flight phase. These changes clearly indicate how the neuromuscular adaptation progressed to achieve constant work output during SSC exercise. Only minor kinematic alterations have been reported in the other type of SSC action such as running, in which about 5 % alteration has been seen [13, 14]. Therefore, the different magnitude of the kinematic alterations in the different type of the SSC action seems to be characterized by the task dependent fatigue response as suggested before [16].

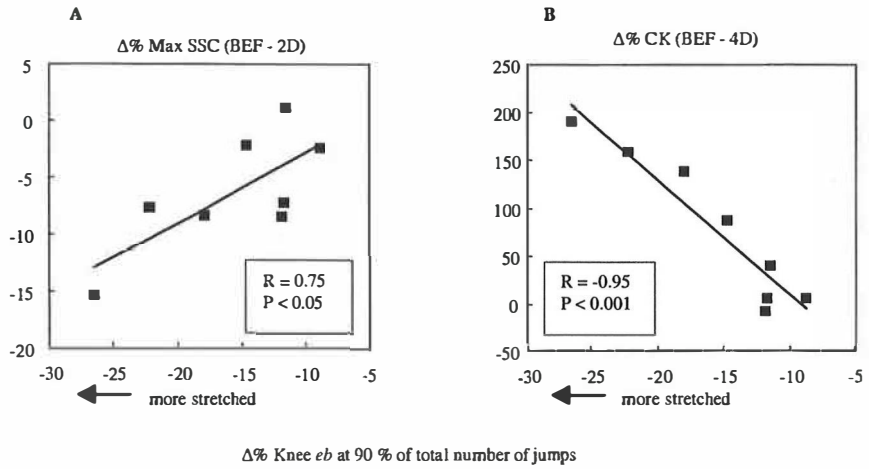

FIGURE 6 Correlation between changes (Δ) in Knee *eb* at the prior to the exhaustion (90 % of total number of jumps) and SSC performance 2 days after the exercise (A) and serum creatine kinase (CK) activity 4 days after the exercise (B).

As shown in Fig. 3, fairly stable Fz *eb* until middle stage of the exercise (60 % of the total jump series) showed that the force output during the braking phase remained constant until the second half of the exercise. On the other hand, Knee *eb* decreased progressively immediately after the start of the exercise indicating increased muscle length of the knee extensors (Fig. 3 B). As the force production is the muscle length dependent [e.g. 34], the longer muscle lengths and/or sarcomere lengths (descending limb) induce decrease in force output [10, 19, 20]. Consequently, continuous decrease in Knee *eb* observed in the present study may induce depression of the force output during braking phase of the SSC exercise. Some adaptation process would be required to maintain constant Fz *eb* until middle stage of the exercise. In this regard, it seems worthwhile to note that EMG activities of the knee extensors during braking phase increased linearly until middle stage of the exercise (Fig. 4). Therefore, eccentric EMG activities of the knee extensors might compensate for the loss of force generation capacity due to the increased muscle length during braking phase of the SSC action. Thus the eccentric part of the SSC cycle seemed to operate effectively only until the end of middle stage of the exercise. Change in the contact time was also stable after the initial small increase until 60 % of the total number of jumps as well as changes in the Knee and Ankle ROM. These small alterations in the kinematics would indicate that the neuromuscular strategy, instead of the movement alterations, is dominating during the braking phase until the end of middle stage of the exercise, which corresponds to the region 1 category of the adaptation process proposed by Patla [33].

During the second half of the exercise, large amount of alterations were seen in both kinematics and kinetics of SSC action (Fig. 3). Continuous decrease in Fz *eb* after the middle stage of the exercise implies the loss of force production during braking phase due to the almost plateau level of eccentric EMG activities of the knee extensors despite the increased absolute muscle length of the knee extensors. These interactions suggest the dysfunction of the eccentric part of the SSC action. It might be supposed, therefore, that the adaptation process could occur during push-off phase rather than

braking phase in the second half of the SSC exercise as the knee extensors EMG activities increased continuously during the push-off phase (Fig. 4). However, the increase in VM EMG activity (≈10 %) was much less than that observed in VL EMG activity (≈20 %). In addition, the triceps surae muscle activities decreased during the corresponding period especially in the GA muscle. These EMG modifications suggest the heavier reliance on the VL muscle during the push-off phase in the second half of the exercise. In line with the large modulations in the contact time and Fz *eb*, Knee ROM increased progressively after the middle stage of the exercise while that of the Ankle ROM decreased. Thus the knee joint further compensated the ankle joint deficiency by increasing the range of motion, resulting in increase in the contact time. This symmetrical relationship between knee and ankle joints would imply the trade-off mechanism between muscles across the joints [8, 33]. Consequently, alterations in movement pattern as well as neuromuscular strategy seem to have dominated during second half of the exercise, which are categorized as region 2 of the adaptation process [33]. The turning point between region 1 and 2 might have existed at 60 % of the total number of jumps in which approximately 90 seconds elapsed in the present experiment.

In the present study, different patterns of synergist EMG activities could be seen between braking and push-off phases in each knee and ankle extensors (Fig. 4). In the braking phase, parallel synergist EMG activities were seen during the whole time course of the exercise. This implies the trade-off mechanism across the joints without any compensation within synergist muscles. On the other hand, a larger increase in VL EMG activity as well as a smaller decrease in SO EMG activity compared to the other synergist activity in the push-off phase would reveal that the adaptation process includes not only trade-offs across the joints but also compensation within the synergist muscles. Thus, the neuromuscular adaptation process might be different between the braking phase and push-off phases of the SSC action. In addition, uniform adaptation pattern observed in each phase during the whole time course of the repeated SSC exercise seems to suggest stable neuromuscular adaptation patterns in each phases without any definite alteration during the course of the exercise. This may imply firm interaction within the synergist muscles in each phase. These adaptation processes could be explained partly by a unique activation strategy in the eccentric action [15]. In addition, continuous depression in triceps surae EMGs during the whole SSC action might suggest greater susceptibility of the ankle joint to the SSC induced fatigue.

In the flight phase, changes in the pre-activities of the EMGs revealed different patterns as compared to those observed in the contact period of SSC action. In short, acute common depressions were seen at the initial stage of the exercise (20 % of total number of jumps) in four muscle groups followed by individual wide variability of muscle activities until exhaustion. SO and VL EMGs showed smaller depressions as compared to those in the GA and VM EMGs. Joint kinematics also changed in greater magnitude, and 10 – 20 fold larger alterations (100 % - 200 %) were seen as compared to those in the contact phase (10 – 20 %). These alterations seem to suggest greater susceptibility for changes in the pre-landing activities as compared to the post-landing activities. It has been argued that the pre-landing EMG activities and/or postural adjustments before contact as shown in running, hopping as well as drop landing are programmed to perform efficient movement or to prevent injuries during subsequent contact period [11, 12, 29, 35]. The kinematic and EMG alterations in the flight phase during SSC exercise would therefore imply possible modifications of the motor control strategy involving the central nervous system to maintain the repeated SSC actions at constant performance level (jump height). In the second half of the exercise (70 % of

the total number of jumps), relative decrease in VL pre-activity and increase in ankle flexion velocity before landing were related with the increase in Knee ROM during contact period (Fig. 5). Increased Knee ROM was further related with the increased contact time. Increased ankle flexion velocity that corresponds to dorsiflexion before landing would seem to reduce the damping capacity of the ankle joint in the subsequent contact period. Thus, the knee joint may have compensated the ankle joint function by the increasing range of motion. This trade-off mechanism as well as those in the EMG activities might suggest that the alteration in the ankle joint function may be the trigger of the decrement of the SSC performance. Decreased VL pre-activity would also induce inefficient landing activity in the braking phase. Consequently, these pre-landing factors seem to affect the braking phase of the SSC action and finally influence the over all performance of SSC as revealed by the increased contact time. These interactions further imply possible interference of the central adaptation during the second half of the fatiguing SSC exercise.

It has been shown that eccentric action as well as SSC action induces delayed muscle damage and pain [21, 27]. The precise mechanism of muscle damage seem to be rather complicated. Strain rate, external work done, force level, duration of stretch, and number of stretches could influence the magnitude of initial mechanical injury [9, 28] and probably the subsequent secondary injury accompanied with the delayed inflammatory response [1]. Recent studies have revealed additional information that the final muscle length during eccentric contraction can influence the initial injury and subsequent delayed muscle damage in the mouse muscle in vitro [23] as well as in the human muscle in vivo [10]. Hunter and Faulkner [23] have shown that the final muscle length is a single best predictor of the force deficit rather than relative displacement and/or initial length of the muscle. Child et al. [10] have also confirmed that the longer final muscle length, in which the range of motion is kept at same level, induces greater force deficit and delayed greater increase in CK activities. This implies susceptibility to greater muscle damage at greater muscle lengths as compared to shorter muscle lengths in the knee extensor muscles. In line with these observations and suggestions, relative increase in final muscle length of the knee extensors during braking phase of the SSC action (Knee *eb*) at the prior to exhaustion (90 % of total jump series) was related with the relative delayed increase in CK activities and with the relative delayed decrease in SSC performance (Fig. 6). In the present study, the knee extensors were subjected to continuous increase in muscle length from the initial stage as observed in the progressive decline in Knee *eb* (Fig. 3). Thus, present results imply that the cumulative lengthening effect during SSC exercise could affect the post-exercise delayed muscle damage and performance deficit.

In conclusion, neuromuscular adaptation process could be clearly modified during exhausting SSC exercise. Adaptation process seemed to consist of neuromuscular adaptation in the braking phase at the first half of the repeated SSCs followed by neuromuscular as well as movement alterations in the push-off phase at the second half of the repeated SSCs. In the second half of the exercise, pre-landing activities, especially in the ankle joint, have significant role in regulating the subsequent kinematics and performance during contact period. Within each part of the SSC action, different adaptation processes might be operative between braking phase and push-off phase. These unique adaptation patterns finally may interact with the post-exercise performance and delayed muscle damage.

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VI

Changes in neuromuscular performance during repeated stretch-shortening cycle (SSC) exercise II: interaction between joint kinematics and time-varying stiffness regulation

by

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(submitted)

CHANGES IN NEUROMUSCULAR PERFORMANCE DURING REPEATED STRETCH-SHORTENING CYCLE (SSC) EXERCISE II: INTERACTION BETWEEN JOINT KINEMATICS AND TIME-VARYING STIFFNESS REGULATION

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Abstract

Repeated and exhaustive stretch-shortening cycle (SSC) exercise was carried out to investigate how the pre-landing motor control and post-landing stiffness regulation are interrelated. For this purpose, time-varying stiffness measurement was employed using the mass spring damper system. Repeated SSC exercise on a special sledge ergometer induced clear alterations in the adaptation of the neuromuscular system as revealed by changes in joint kinematics and surface electromyographic activities. The results revealed the interaction between increased ankle flexion velocity and decrease in initial leg stiffness at the latter part of the exhaustive exercise ($r = -0.75$, $P < 0.05$). The decreased initial leg stiffness was negatively related with the increase in contact time ($r = -0.89$, $P < 0.01$) and viscous ratio at the end of braking phase ($r = -0.75$, $P < 0.05$). This implies that the decrease in initial leg stiffness induces additional performance decrement and increase in viscosity at the end of eccentric phase of the SSC action. Increased viscous ratio showed positive relationship with the delayed increase in serum CK activity, an indirect marker of muscle damage ($r = 0.83$, $P < 0.01$), suggesting that the initial muscle injury influences the leg viscosity. Thus, repeated and an exhaustive SSC exercise induces alterations of performance strategy including pre-landing activities that affect post-landing initial leg stiffness during latter part of the time course of exercise.

1. Introduction

The stretch-shortening cycle (SSC) of muscle action is characterized by the active stretch (eccentric action) followed immediately by the concentric action [e.g. 23]. The purpose of the SSC is to produce greater work output during the concentric phase as compared to pure isometric and concentric action. This may be due to higher force level at the starting point of the concentric action attained by active stretch during eccentric phase [e.g. 29, 30] or to the reuse of the stored elastic energy during the concentric phase [11]. In addition when the active muscle is forcibly stretched, the stretch reflex plays an important role in increasing the muscle stiffness and to counteract the force yielding during the braking phase [1, 16, 26]. Reflex potentiation is presumably associated with both improvement of stiffness and increase in elastic energy during SSC action [7, 24]. Thus stiffness regulation certainly has an additional important factor to perform SSC action more effectively.

It has been shown recently that the exhaustive repeated SSC exercises of either short duration with rebound jumps [27, 18, 19] or longer lasting marathon running [2, 3] induces similar disturbances in the stiffness regulation. Nicol et al [27] were the first ones to report a reduction in stretch reflex amplitude after exhaustive short term SSC exercise. This was followed by Avela and Komi [2] who demonstrated the reduced short latency stretch reflex accompanied with the reduction of peak stiffness in the soleus muscle during sledge jumps tested after the marathon running. Similarly, Horita et al. [18] have shown the interrelationship between decrease in short latency stretch reflex of the vastus lateralis muscle, knee joint angular stiffness and knee joint peak power during drop jump tested after the short term repeated SSC exercise lasting approximately 3 min. Furthermore these studies have observed similar delayed possible muscle damage as judged indirectly by the delayed increase in serum creatine kinase (CK) activity. Therefore it has been suggested that the reduced stiffness regulation after the SSC

exercise could be partly associated with the reduced reflex sensitivity via presynaptic inhibition from the group III and IV afferents induced by the delayed muscle damage occurring in both extrafusal and intrafusal fibres [3, 27].

In addition to these possible changes of neural input, delayed alterations in pre-landing kinematics can also have influence on the subsequent post-landing stiffness during the drop jumps tested after the short term exhaustive SSC exercise [19]. Stiffness can be considered as the controlled parameter regulated by muscle length change, activation level and feed back signal from peripheral receptors [20]. It could therefore be proposed that alterations in stiffness could be also observed during repeated SSC exercises in which the strategy of neuromuscular and movement control may change so that the exercise can continue longer [17]. Benz et al. [6] have shown that the decrement of the stiffness is accompanied by the force deficit during repeated eccentric actions by the rabbit muscle model. In a previous paper, we have shown that there may be an interaction between pre-landing kinematics and post-landing performance with subsequent decrement in the post-exercise performance [17]. However, no previous attempt has been made to examine possible influence of the pre-landing activities on the stiffness regulation during natural type of human SSC action. Consequently the present study was undertaken to analyze further the results of the previous paper and to find out the possible interaction between pre-landing activities and post-landing stiffness regulation during repeated SSC actions.

2. Methods

With regard to the details of the methods, reference is made to our preceding paper [17]. For clarity, some of the important methodological aspects are presented here.

The subjects (n=8) performed a series of exhaustive bilateral submaximal rebound jumps along the gliding track of the sledge. The subjects were instructed to rebound as long as possible to a height which was set at 70 % of their pre-determined maximal height. In the present study, the average dropping height was 64 ± 6 cm. The exercise session was stopped when the subject could not reach the submaximal rebound height. On average, the subjects performed 87 ± 24 repetitions, which corresponded to a duration of 2.5 ± 0.7 minutes. To examine the fatigue effect on SSC muscle function, a jump test (Max SSC) was performed on the sledge apparatus before and after the SSC fatigue exercise as well as 2 days and 4 days after. Blood samples were also taken in each test session to measure the lactate (LA) concentration and creatine kinase (CK) activity.

Surface electromyographic (EMG) activities from the vastus lateralis (VL), vastus medialis (VM), medial gastrocnemius (GA) and soleus (SO) muscles of the right leg were recorded during fatiguing exercise and jump test. The EMG signals were stored simultaneously with the force signal as well as goniometric signals recorded from the knee and ankle joint on a computer via a real time data acquisition system (Codas, Dataq Instruments Inc., USA), which included a 12-bit A/D converter with a sampling frequency of 1 kHz. EMG was integrated and then time normalized to calculate average EMG activity in the following three time periods; pre-activation before contact, braking phase (eccentric) and subsequent push-off phase (concentric). The pre-activation phase was defined as 100 ms preceding the ground contact [25].

In the ground reaction force curve of rebound jump, the initial shoulder of force (Fz *init*), the first peak (Fz 1^{st}), and the force level at the end of braking (eccentric) phase (Fz *eb*) were analyzed. In the knee joint kinematics, the peak flexion angle during

flight period, the range of motion during braking phase (Knee ROM) and the angular displacement at the end of braking phase (Knee *eb*) were analyzed. In the ankle joint, pre-landing angular velocity during downward period of the flight phase and range of motion during braking phase (Ankle ROM) were analyzed. The average curves in ground reaction force and joint angles during the course of SSC exercise were shown in Fig. 1. In the present study, the whole time course of the SSC exercise was relatively divided into ten stages (initial stage, 10% – last stage, 100%) based on the individual total number of jumps achieved during exercise. Thereafter, ten successive jump records were pooled in each subject (n=8), in total 80 samples, for calculation of the average curve at each stage of the SSC exercise.

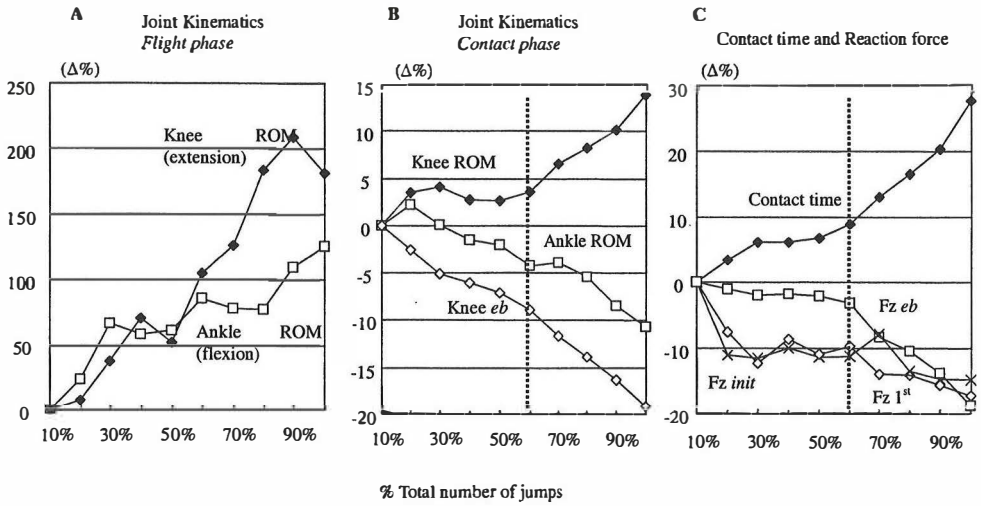

FIGURE 1 Average curves of the knee joint angle (*top*), ankle joint angle (***middle***) and reaction force (*bottom*) in each stage of the stretch-shortening cycle (SSC) exercise (*bold line*, initial stage; ***thin line with circle dot***, middle stage; ***thin line***, last stage). Contact period normalized as 100 %. Flight period normalized as –100 %. In the reaction force, the initial shoulder (Fz *init*), first peak (Fz 1st) and force level at the end of braking phase (Fz *eb*) are denoted as circles.

2.1. Identification of time-varying stiffness during SSC action

In the present study, the lower leg was modeled as simple mass spring damper system described elsewhere [12, 30, 31]. Equation of the motion of the system as a function of the time was as follows:

$$F(t) = M\ddot{x}(t) + c\dot{x}(t) + kx(t) \quad \text{---------- (1)}$$

where F is the reaction force, M the mass of the subject and the gliding part of the sledge, c the leg viscosity, k the leg stiffness and x the displacement of the sledge sensed by rotary encoder attached to the gliding part of the sledge. This model represents the total leg system as the combination of three lower limb joints (hip, knee and ankle) and does not distinguish reflex components and intrinsic muscle components. Stiffness has

been considered as the controlled parameter regulated basically by the length feedback component originating from the spindle discharge and by the force feedback component originating from the tendon organ discharge as well as by the muscular component [20]. Thus stiffness would be regulated instantaneously for load compensation in the natural circumstances such as SSC action. Therefore, the time-varying stiffness (TVS) was analyzed every 1 ms during contact period of the SSC action by using the time-varying moving correlation method that modifies the original method described by Bennett [4, 5]. The precise description is as follows: In the equation (1), F, M, $\ddot{x}, \dot{x}, x$ are known. Thus equation (1) can be rearranged as equation (2) as follows:

$$F(t) - M\ddot{x}(t) = c\dot{x}(t) + kx(t) \quad \text{---------- (2)}$$

When analyzing the corresponding time, (*t*)th data, the variance-covariance matrix was created by using the three sequential data sets of equation (2) (3 ms duration) consisting of the (*t-1*)th, (*t*)th and (*t+1*)th data to avoid phase shift of the estimated parameters. Within an interval the parameter c and k were assumed to be constant and then estimated with conventional linear multiple correlation technique. It could be supposed that initial impact shock may contaminate the estimated parameters. However, inertial force (mass × acceleration) was subtracted in the left hand side of the equation (2). The remaining part of the reaction force might represent the pure muscle-tendon force. Thus, the initial impact component is negligible in this approach. These procedures were repeated every 1 ms during contact period of the SSC action during exercise. After the calculation, the average curves of the stiffness and viscosity were constructed by the same method used in kinetics and kinematics analysis.

2.2. Significant consideration of TVS for assessment of the stiffness regulation during SSC action

An example of the results obtained from one subject is presented in Fig. 2. There were two distinguishable peaks in the TVS. The stiffness first peak (Stiff 1st) appeared within 10 ms after the contact, which corresponded to initial rising phase of the reaction force to the initial shoulder. At point of the Stiff 1st, the ankle joint flexed only 0.3 deg whereas the knee joint in which the knee joint flexed 1.3 deg. It has been suggested further that the initial shoulder of the tension is due to the stretch of the elastic elements including the attached cross bridges [32]. In the present example, the initial shoulder occurred at 2.6 deg of flex in the ankle joint. This is comparable with the data obtained from relaxed human ankle extensor muscle-tendon complex in which the steep rise in tension appeared during the first 1 – 2 deg of flex in the ankle joint [21]. On the other hand, the knee joint already flexed 3.7 deg at the occurrence of the initial shoulder. Thus Stiff 1st and the initial shoulder are components which refer to the initial resistance of the ankle joint to the initial impact, and may also characterize the short range stiffness of the ankle extensors [28]. After the initial shoulder, the ankle joint flexion occurred simultaneously with the decrease in TVS and reaction force that implies partly the first break down of the attachment between actin and myosin filament beyond overlap within the sarcomere as observed in the striated animal muscle [13] as well as in the human muscle [21]. It has also been shown that the initial shoulder of the reaction force could be modified by the landing styles, either the heel contact or the metatarsal contact [14]. However, in the present study, all the subjects performed similar landing style in which

the metatarsal to heel contact was observed. Therefore, the effect of landing style on the reaction force can be neglected. Consequently, the muscular component rather than the force feed back and length feed back components might dominate in the over all stiffness regulation during the initial rising part of the reaction force. Especially relevant in this case is the observed small length changes in the ankle and knee extensor muscles.

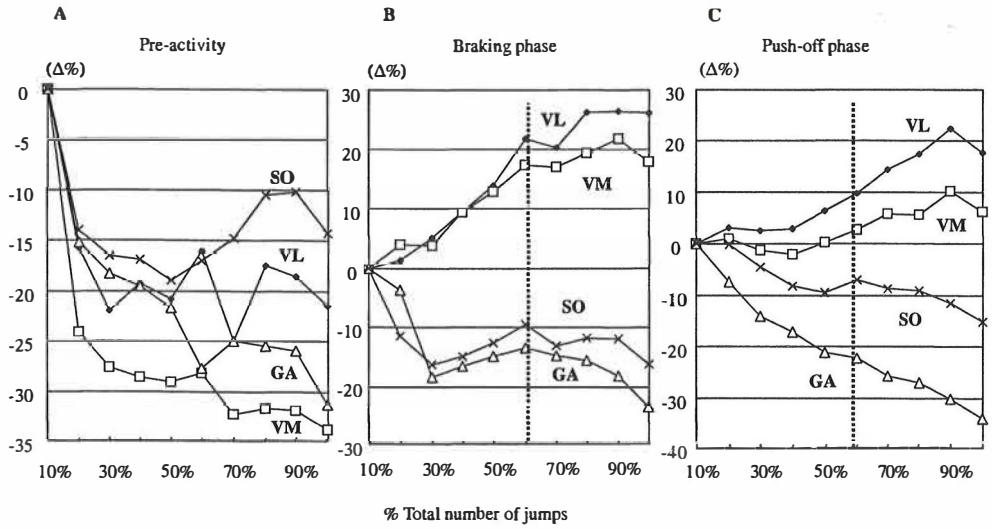

FIGURE 2 Records of the vertical reaction force (Fz, *top*), ankle and knee joint ankle (*top*), time-varying leg stiffness (*middle*) and time-varying leg viscosity (*bottom*) of the single SSC action in one subject. Small deflection point of the knee joint angle is denoted as eclipse. Some Stiffness and viscous parameters also denoted as circles.

Stiffness second peak (Stiff 2nd) was seen during the secondary rising phase of the reaction force, timed with Fz 1st after the initial shoulder. After the initial shoulder of the reaction force, TVS showed acute depression followed by secondary increase that implies partly reattachment of the cross-bridges. At this point, both the knee and ankle joint angles changed implying involvement of the length feed back component of the over all stiffness regulation. Stiff 2nd appearing before the Fz 1st corresponds to the small deflection point of the knee joint angle change (denoted as eclipse in the Fig. 2). Because the muscle length as well as stiffness have been considered as regulated variables by the motor servo [20], it seems logical that Stiff 2nd appeared in phase with the knee joint angle change. Thus Stiff 2nd might be mainly related with the knee joint function in which the muscular and length feed back components are involved. After the Stiff 2nd, TVS already started the decrease before Fz 1st during lengthening phase. This implies that the negative force feed back component originating from the tendon organ discharge would overcome the length feed back component in the over all stiffness regulation. As a consequence during the second rising part of the reaction force, the knee extensor muscles seemed to be involved mainly in the over all stiffness regulation in which muscular, length feed back and force feed back components might be operative. As shown by the more restricted condition [5], time-varying viscosity (TVV) showed symmetrical pattern with the TVS. In addition, the absolute values of both TVS peaks ranged between 15 – 30 kN·m^{-1}, which were comparable within a range of the

data for lower body musculotendinous stiffness using the same model with different analytical procedures reported elsewhere [31]. Consequently, TVS approach accompanied with the joint angle change could give information about the stiffness regulation. Therefore Stiff 1st, Stiff 2nd, stiffness at the end of braking phase (Stiff *eb*) were analyzed in the present study in addition to the first peak (Vis 1st) as well as second peak (Vis 2nd) of the TVV. The average curves of TVS and TVV at the each stage during SSC exercise are shown in Fig. 3.

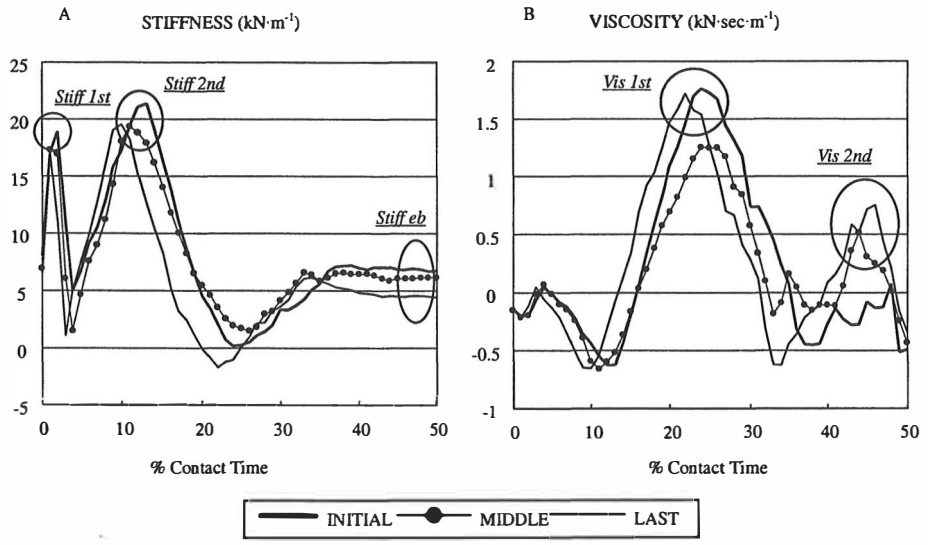

FIGURE 3 Average curves of the time-varying leg stiffness (A) and time-varying leg viscosity (B) during braking phase of the SSC exercise in each stage of the exercise. The time axis is normalized in the same way as in Fig. 1. The stiffness and viscous parameters also denoted as circles.

2.3. Blood analysis

The blood lactate concentration was determined at rest, immediately after the sledge exercise as well as 3, 5 and 30 min later using a lactate analyzer (model 640, Roche). The serum CK activity was measured at rest and 30 min after the sledge exercise as well as 2 days and 4 days after exercise. The CK activity was analyzed using a CK ultra-violet test kit (Boehringer Mannheim, Germany).

2.4. Statistics

Time varying changes of the parameters during exercise were tested by an analysis of variance (ANOVA) for repeated measurements on one factor and then a post-hoc LSD multiple comparison was performed. Pearson's correlation coefficients were calculated to examine the relationship between the relative changes of the different parameters. Statistical calculations were performed with a statistical package (STATISTICA ver. 4.5, StatSoft, USA). The level of significance was set at $P < 0.05$.

3. Results

The basic results were already mentioned in our preceding paper [17]. Therefore they are described only briefly here.

3.1. Max SSC performance, Blood LA and CK changes

Blood LA concentration increased at 5 min after the SSC exercise (7.1 ± 0.8 $mmol \cdot l^{-1}$, $P < 0.001$) as compared to before exercise (1.5 ± 0.3 $mmol \cdot l^{-1}$). Serum CK activity was markedly elevated on the 2nd day (477 ± 272 $U \cdot l^{-1}$, $P < 0.001$) as well as 4th day (373 ± 172 $U \cdot l^{-1}$, $P < 0.05$) compared to pre-exercise levels (211 ± 41 $U \cdot l^{-1}$). Max SSC rebound height decreased on 2nd day (85.8 ± 4.9 cm, $P < 0.01$) as compared to pre-fatigue performance (91.8 ± 8.0 cm). Therefore the present SSC exercise induced acute metabolic fatigue and delayed possible muscle damage accompanied with the delayed decrease in SSC performance.

3.2. Changes in kinetics, kinematics and EMG activities during repeated SSC exercise

Relative changes in kinetics, kinematics and EMG activities during repeated SSC exercise are shown in Fig. 4 and 5. During first half of the exercise (≤60 % of total number of jumps), contact time and Fz *eb* showed minor changes accompanied with the similar small changes of the knee and ankle ROM. Corresponding increase in EMG activities of the knee extensor muscles during braking phase indicates compensation of the knee extensors during braking phase. Thus the braking phase of the SSC action was only slightly influenced during the first half of the exercise. In the second half of the exercise, (≥60 % of total number of jumps), the contact time increased linearly accompanied with the linear decrease in Fz *eb*. Simultaneous increase in Knee ROM as well as decrease in Ankle ROM were observed. Decrease in Fz *eb* seems to suggest depression of force output during braking phase that was confirmed by the no additional increase in knee extensors EMG activities. Decreased force output was also found in the smaller average force-time curve at the last stage of the exercise (Fig. 1). Symmetrical relationship between Ankle ROM and Knee ROM indicated the trade-off mechanism across the joints. In addition, increase in EMG activities of the knee extensors during push-off phase suggested the heavier reliance of the knee extensors especially in the VL muscle. As a consequence, the braking phase of the SSC action was depressed during the second half of exercise that implies the possible compensation occurred in the push-off phase.

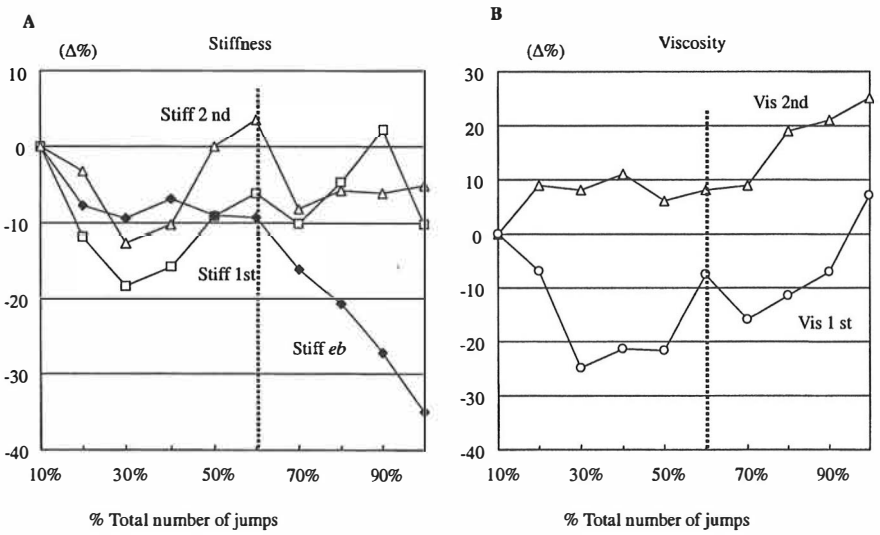

FIGURE 4 Relative changes in kinetics and kinematics during the time course of the SSC exercise. A: joint kinematics during flight phase. B: joint kinematics during contact phase. C: contact time and vertical reaction force. ROM; range of motion. Knee *eb*: knee joint angle at the end of braking phase. Fz *init*: initial shoulder. Fz *eb*: force level at the end of braking phase. Fz 1st: first peak of reaction force. Horizontal axis indicates the total number of jumps as relative values.

In the flight phase, joint kinematics changed considerably, in which 10 – 20 fold larger alterations were found as compared to those observed in the contact phase. Average curves of the kinematics also showed the greater alterations (Fig. 1). Increased ankle flexion ROM during flight period was related to increased ankle flexion velocity before landing. EMG activities also showed larger alterations as compared to post-landing EMG activities. These alterations indicate great sensitivity of the neuromuscular adaptation process during the pre-landing period.

3.3. Changes in TVS and TVV during repeated SSC exercise

Relative changes in the selected parameters of TVS and TVV during SSC exercise are shown in Fig. 6. Stiff 1st changed from 21 ± 8 $kN \cdot m^{-1}$ (initial stage) to 18 ± 7 $kN \cdot m^{-1}$ (last stage, N.S.). Stiff 2nd changed from 24 ± 9 $kN \cdot m^{-1}$ (initial stage) to 22 ± 5 $kN \cdot m^{-1}$ (last stage, N.S.). Both Stiff 1st and Stiff 2nd showed similar U shaped change during the first half of the exercise. In contrast, Stiff *eb* decreased at the last stage of the exercise (4 ± 1 $kN \cdot m^{-1}$) as compared to the initial stage (7 ± 3 $kN \cdot m^{-1}$, $P < 0.001$). Change in Stiff *eb* was similar to those observed in Fz *eb* characterized by the small change during the first half of the exercise followed by the linear progressive decrease during the second half of exercise. Vis 1st decreased at the first half of the exercise (30 – 50 % of total number of jumps, $P < 0.01$) with the similar manner seen in the Stiff 1st and Stiff 2nd changes during the first half of the exercise. Vis 2nd increased at the last stage of the exercise (0.7 ± 1 $kN \cdot sec \cdot m^{-1}$) from the initial stage (1.7 ± 0.9 $kN \cdot sec \cdot m^{-1}$, $P < 0.001$). Change in Vis 2nd showed similar pattern to those observed in the contact time. As

shown in Fig. 3, pronounced increase in Vis 2nd was shown as compared to Vis 1st at the last stage of the exercise. Thus the ratio between Vis 2nd to Vis 1st (Viscous ratio) increased from 0.43 ± 0.26 (initial stage) to 0.75 ± 0.28 (last stage, $P < 0.001$).

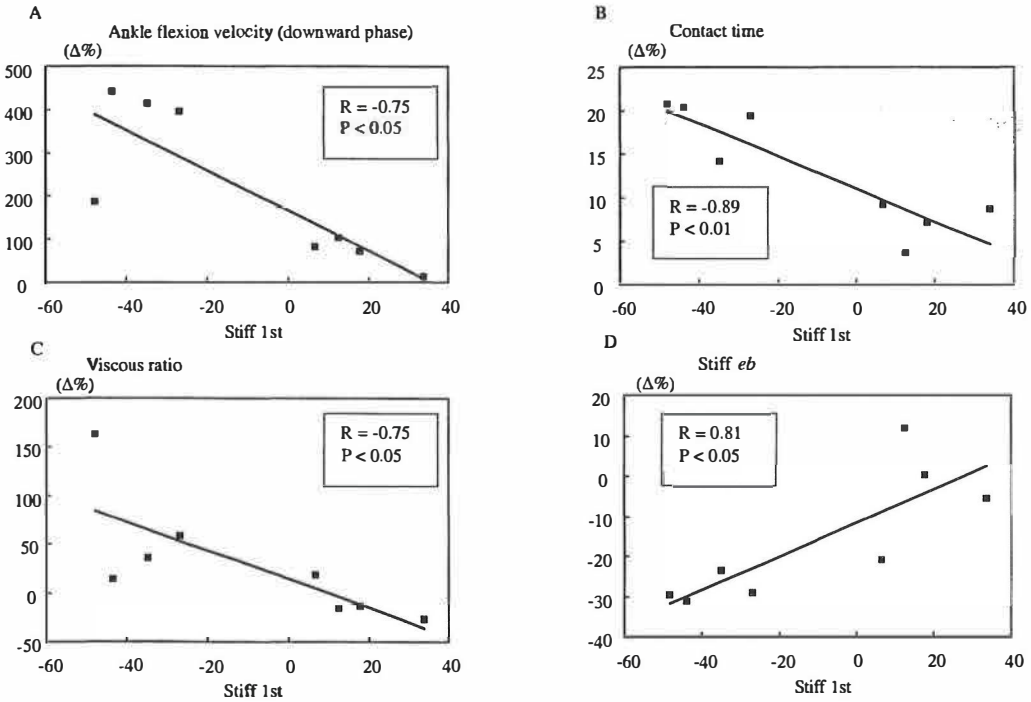

FIGURE 5 Relative changes in EMG activities of the VL, vastus medialis (VM), GA and soleus (SO) muscles during time course of the SSC exercise. A: pre-activity. B: braking phase. C: push-off phase. Horizontal axis has the same scale as in Fig. 4.

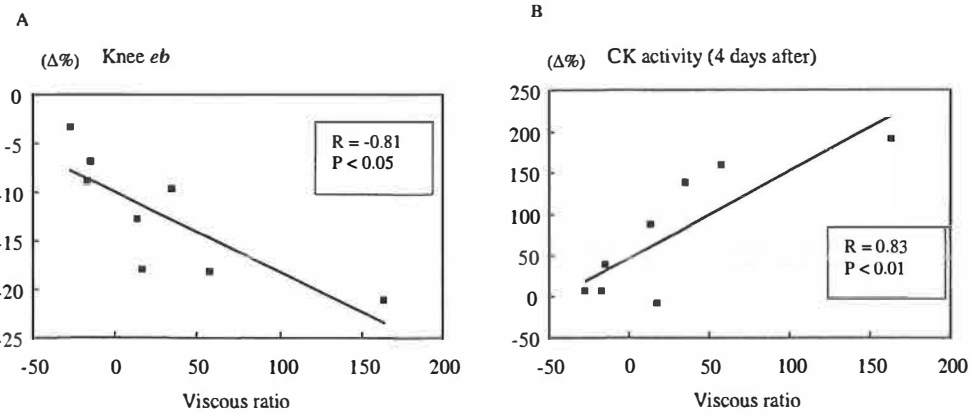

FIGURE 6 Relative changes in stiffness parameters (A) and viscous parameters (B) during the time course of the SSC exercise. Stiff 1st: first peak of stiffness. Stiff 2nd: second peak of stiffness. Stiff *eb*: stiffness at the end of braking phase. Vis 1st: first peak of viscosity. Vis 2nd: second peak of viscosity. Horizontal axis has the same scale as in Fig. 4.

3.4. Relationships between viscoelastic parameters and kinematic, mechanical and biochemical parameters

In our preceding paper, relationship was found between alterations in pre-landing activities and post-landing performance during the second half of the SSC exercise in addition to post-exercise performance decrement [17]. In the present analysis, relative decrease in Stiff 1^{st} was related with the relative increase in the pre-landing ankle flexion velocity ($P < 0.05$), contact time ($P < 0.01$) and viscous ratio ($P < 0.05$) as well as with decrease in Stiff *eb* ($P < 0.05$) at the second half of the exercise (70 % of the total number of jumps, Fig. 7). Increased viscous ratio at the second half of the exercise (70 % of the total number of jumps) was further related with the decrease in the Knee *eb* ($P < 0.05$) and delayed increase in CK activity 4 days after the exercise ($P < 0.01$, Fig. 8).

4. Discussion

In the preceding paper [17], we have demonstrated the significant interaction between pre-landing activity and post-landing performance at the second half of the SSC exercise. In the second half of the exercise, Knee ROM and contact time increased progressively accompanied with the decrease in Fz *eb* and no additional increase in EMG activities of knee extensor muscles during braking phase of SSC action. In addition, pre-landing activities which include ankle flexion velocity and VL EMG activity influenced the post-landing Knee ROM. These results indicates possible influence of neuromuscular strategy during flight phase on the subsequent knee joint function and heavier reliance to the knee joint during second half of the exercise. Because alterations of pre-landing activities induce changes in muscle length and/or activation level it could be hypothesized that these alterations have an influence on the stiffness regulation especially in the initial impact phase of the contact period of SSC action.

To clarify this hypothesis, Stiff 1^{st} and Fz *init* were analyzed in the present paper. As shown in Fig. 7, decreased Stiff 1^{st} at the initial impact phase was related with the increased ankle flexion velocity before landing at the second half of the exercise. As already discussed (*see* Methods), Stiff 1^{st} might be indicative of the initial attachment of the cross bridges (short range stiffness). Forcefully stretched ankle extensors due to the increased ankle flexion velocity before landing may likely induce detachment of the cross bridges beyond the over lap in some of the weakest sarcomeres at the landing and this could have caused the decrease in the Stiff 1^{st}. In the present study, Fz *init* decreased continuously during exercise (initial stage, 0.65 ± 0.1 kN; last stage, 0.54 ± 0.43 kN, $P < 0.001$, Fig. 4). Wood et al. [32] have observed similar phenomenon during repeated eccentric actions of the frog muscle in vitro. These authors considered that the initial shoulder of the tension rise was due to the stretch of the attached cross bridges in which some of the weakest sarcomeres are over stretched because of the heterogeneity in the sarcomere length along the fibres [9]. The present results further give additional suggestions of the possible influence of motor strategy, which involves the pre-landing activities that may alter the sarcomere kinetics, on the subsequent immediate post-landing stiffness during repeated SSC actions by humans.

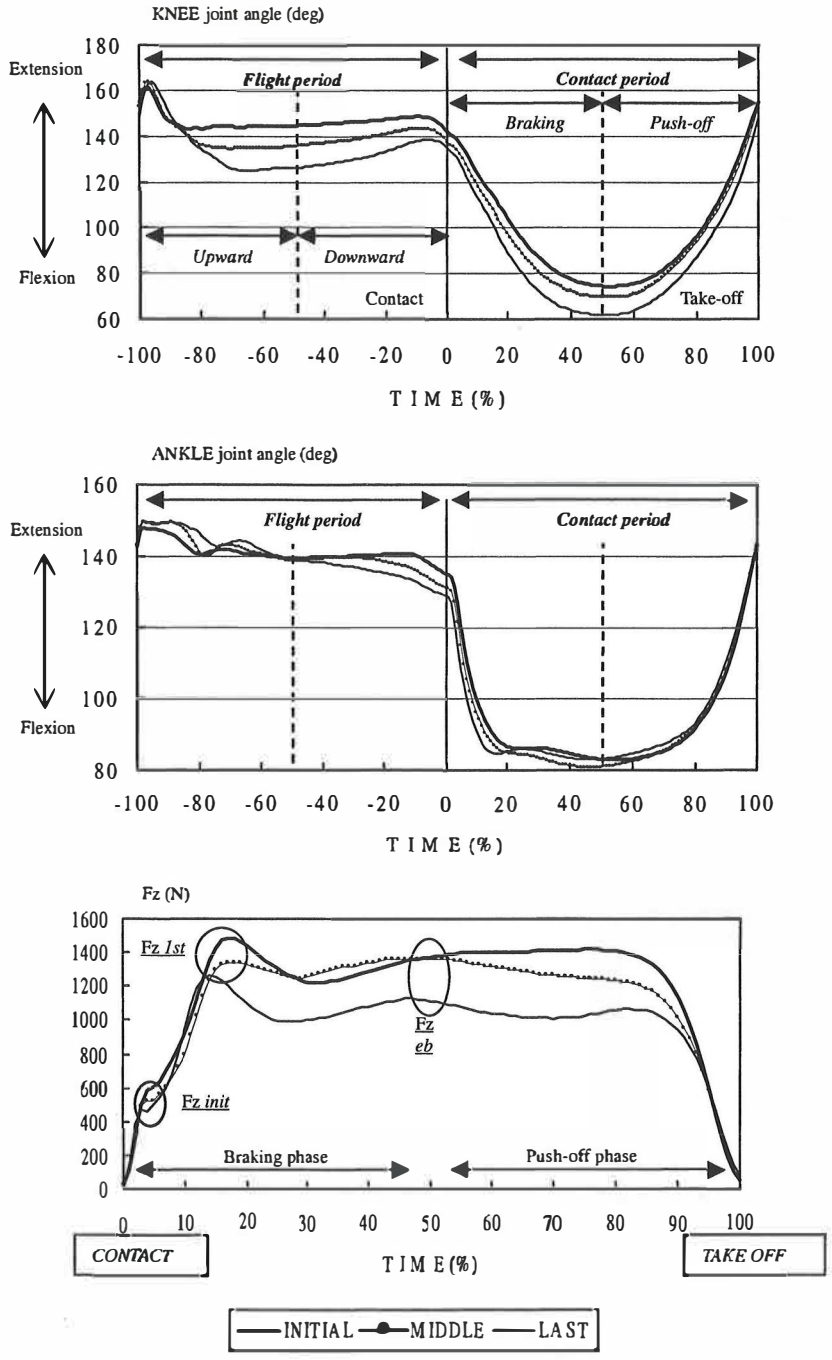

FIGURE 7 Correlation between changes (Δ) in Stiff 1st and ankle flexion velocity during downward phase (A), contact time (B), viscous ratio (C) and Stiff *eb* (D). All values were collected at second half of the SSC exercise (70 % of total number of jumps).

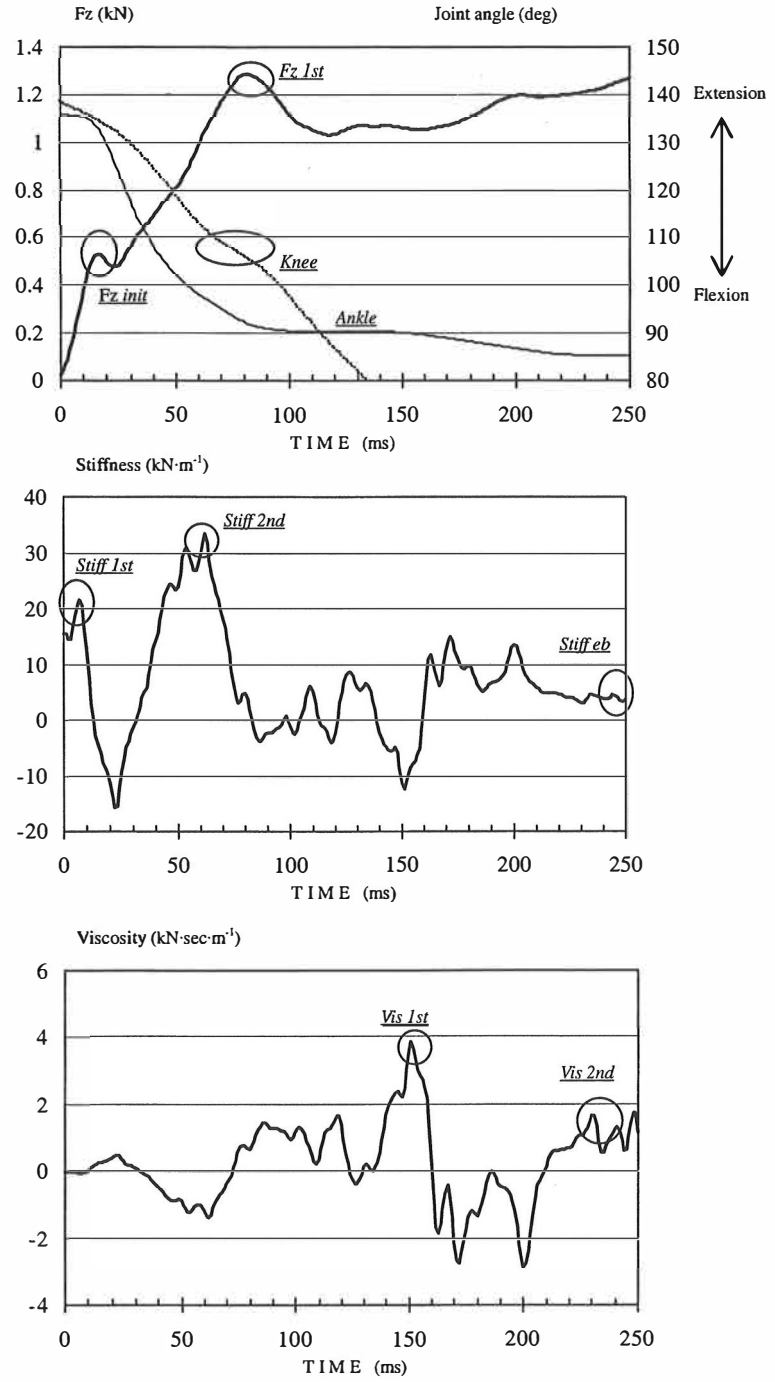

FIGURE 8 Correlation between changes (Δ) in viscous ratio and knee joint angle at the end of braking phase (Knee *eb*, A) and CK activity at the 4 days after the SSC exercise (B). Kinematic and viscous values were collected at second half of the SSC exercise (70 % of total number of jumps).

Decrease in Stiff 1st was further related to the increase in contact time, viscous ratio as well as decrease in Stiff *eb* at the second half of the exercise. Decrease in Stiff *eb* was also related with the decrease in Fz *eb* ($r = 0.74$, $P < 0.05$) that indicates the decrease in force output during braking phase. In addition, increase in the viscous ratio that was related with the decrease in Knee *eb* ($r = -0.81$, $P < 0.05$, Fig. 8) indicates increase in the knee extensor muscle length during braking phase. These additional relationships imply further that the initial stiffness decrement may influence the subsequent stiffness regulation and performance changes during contact period. Stiff 1st seems to indicate mainly muscle stiffness of the ankle extensors (*see* Methods). It can, therefore, be suggested that the depressed ankle extensor properties at landing, which partly resulted from pre-landing activities, might be a trigger for the subsequent performance decrement at this stage. Our previous results also suggested the greater susceptibility of the ankle joint function in terms of continuous decrease in EMG activities and greater alterations in the pre-landing kinematics [17].

Increase in viscous ratio implies that the relative increase in the Vis 2nd was greater than that of Vis 1st (Fig. 3). Because Vis 2nd is observed at the end of braking phase, increase in viscous ratio implies also increase in the viscosity during the turning point from braking to push-off phase. Negative relationship between viscous ratio and Knee *eb* (Fig. 8) suggests possible influence of the knee joint angle on the viscosity at the end of braking phase. Thus increase in leg viscosity might be attributed to the lengthening of the knee extensor muscles. It has been suggested that some of the over stretched weakest sarcomeres due to the heterogeneity of the sarcomere length along the fibres [9] induce fiber injury and then link to the delayed inflammatory response which lead to delayed muscle damage and pain [22, 32]. In addition, increased fibre lengthening and activation may further induce increased heterogeneity in sarcomere length [10]. A recent study has suggested that these mechanisms could be fundamental for initial muscle injury under the wide variability of circumstances involved in daily activities [8]. In the present study, the knee extensors were subjected to continuous increase in muscle length from the initial stage as observed in the progressive decline in Knee *eb* (Fig. 4). Therefore the cumulative lengthening effect would induce possible fibre injury of the knee extensors especially in the second half of the exercise. Higuchi et al. [15] have demonstrated that the thin filament does not re-enter to the thick filament but is buckled at the A-I junction after the extreme stretch. This may interfere with the coupling between stretch and shortening and this phenomenon may be related to increased viscosity at the end of braking phase in the present study. Thus interaction between decrease in Knee *eb*, increase in viscous ratio and delayed increase in CK activity (muscle damage) found in the present study could be accounted for the interference of sarcomere kinetics by the cumulative lengthening effect. Negative relationship between Stiff 1st and viscous ratio (Fig. 7) further support the notion that the depletion of the ankle joint function at the landing induces subsequent increased reliance of the knee joint function as suggested previously [17].

At the first half of the exercise, changes in Stiff 1st and Stiff 2nd showed similar U shaped patterns (Fig. 6). Stiff 1st might be regulated mainly by the muscle component. On the other hand, Stiff 2nd would be regulated by the muscle component together with the length feed back component as discussed previously (*see* Method). Interestingly, change in pre-activity of SO EMG showed similar U shaped pattern (Fig. 5). This result may suggest the possible regulation of Stiff 1st by pre-landing SO muscle activity at the first half of the exercise. In addition, similar pattern in Stiff 2nd would suggest that the muscle component might still have dominated the regulation of Stiff 2nd. However at the

second half of the exercise, Stiff 1st and Stiff 2nd showed different patterns. Stiff 1st showed increasing trend until prior to exhaustion (90 % of the total number of jumps) which is similar to those observed in pre-activity of SO muscle. Stiff 2nd showed a plateau (70 –100 % of total number of jumps). VL and VM EMGs during braking phase also showed a plateau during the second half of the exercise. Stiff 2nd may therefore be affected partly by the knee extensor EMGs of the braking phase. As many of these notions are still speculative, these interactions between EMG and stiffness regulation may require additional and more precise investigation. However, changes in each pattern of Stiff 1st and Stiff 2nd observed during the present exercise would suggest that stiffness regulation mechanisms are different between the first and second halves of the SSC exercise.

In conclusion, exhaustive repeated SSC exercise induces alterations in movement strategy which has consequences to the post-landing initial leg stiffness during the latter part of the time course of exercise. Decline in the initial stiffness induces additional subsequent performance deficit and viscoelastic alterations during contact period and these changes are finally related to delayed occurrence of muscle damage. It could be suggested that the stiffness regulation mechanism may change during the repeated SSC exercise.

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